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PRINCIPLES OF PERIODONTOLOGY: EARLY OBSERVATIONS, EPIDEMIOLOGY AND CLASSIFICATION. REVIEW.

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Abstract
Periodontology has been defined as the scientific study of the periodontium in health and disease. The objective of this overview of principles of periodontology is presentation of current and established concepts. Aspects of the history, classification and epidemiology of the most common periodontal diseases are discussed. People exhibiting periodontal abnormalities, for example impairments of tissue integrity or function, are said to have periodontal diseases. Periodontal diseases are arguably among the most ancient and common infectious diseases affecting humans, leading to permanent destruction of the supporting structures of the dentition and ultimately tooth loss. The new classification is complex and will take time to fully incorporate into patient care globally.
Keywords: *periodontal disease, current and established concepts, epidemiology, classification*

Introduction

Principles of periodontology is presentation of current and established concepts from Dentino A. et al, 2013[1]. The periodontium includes the gingiva, alveolar bone, periodontal ligament and root cementum, i.e. the tissues that support the teeth. The anatomy, histology and physiology of the normal periodontium have been described in great detail elsewhere [2].

They will not be covered in this context. These comprise a variety of phenotypes; defined by clinical signs and symptoms, they constitute the periodontal syndrome [3,4]. The most frequently observed phenotype is inflammation of the gingiva (gingivitis) induced by dental plaque (biofilm), and includes changes in tissue color, volume, temperature, crevicular exudate and bleeding upon gentle provocation with a probe [5]. The clinical signs of biofilm associated gingivitis are reversible when adequate oral hygiene is implemented and maintained [6]. Less prevalent than gingivitis, but still observed in many persons, are the clinical signs of biofilm-associated periodontitis. They include periodontal pockets, attachment loss, bleeding upon probing, and radiographic bone loss [7].

History: Early observations

Periodontal diseases, unlike caries, are not a by product of modern civilization. Manifesting themselves as ante mortem loss of alveolar bone, signs indicative of periodontal pathology were discovered in human specimens attributed to the Middle Pleistocene stage. For

instance, indications of alveolar bone resorption were found in the 640- to 735,000- year-old Mauer mandible (Homo heidelbergensis) in Germany, and in a 169- to 191,000-year-old mandible that was unearthed at the Bau de l"Aubesier, Vaucluse, France [8,9]. Such findings support the theory that periodontal diseases have plagued humans and their phylogenetic ancestors for a very long time. Chinese physicians were probably first to describe signs of periodontal diseases. Diagnoses and treatments were presented in the earliest known textbook of Chinese medicine, Nei Ching, attributed to Huang Di (approximately 2700–2600 BC)[10].

The ancient Egyptian Ebers Papyrus was written approximately 1000 years later (approximately 1550 BC). It is one of the oldest fully preserved medical documents, and contains several passages on remedies to cure conditions such as loose teeth and swollen gums (flesh)[11]. In Ancient India, Sushruta (approximately 6th century BC) illustrated a large number of surgical instruments, and explained 300 surgical procedures in the treatise Samhita. Sushruta taught a holistic approach to medical therapy. In pursuit of his philosophy, he described four periodontal conditions, offering probably the first classification of periodontal diseases[10].

In 1563, the Italian Renaissance anatomist and physician Bartolomeo Eustachi (1514–1574) completed Libellus de Dentibus, the first book dedicated exclusively to the description of teeth [12]. He was the first to describe the periodontal ligament, as well as the deciduous and permanent dentitions. Eustachis profound understanding of head anatomy and his amazing eye for the detail led him to link increased tooth mobility at an advanced age with widening of the space between the root and the alveolar bone. Similarly remarkable, he prescribed the removal of calculus and granulation tissue using scalers and curettes, respectively, to encourage re-attachment of periodontal tissues to loose teeth [13].

From scurvy of the gum to Riggs disease

Almost two centuries after Eustachi, the French surgeon-dentist Pierre Fauchard (1678–1761) published Le Chirurgien Dentiste, a two-volume book dedicated to the practice of dentistry. Fauchards comprehensive work profoundly influenced the practice of dentistry.

He pointed out that any dental disease can be allocated to one of only three classes, namely:

(1) diseases with external cause, (2) (hidden) diseases that attack those tooth parts embedded in the periodontium, and (3) (symptomatic) diseases caused by the teeth [14].

Critical of the theories accepted by most physicians of his time, Fauchard postulated a humoral etiology of periodontal disease that is modulated by local factors, e.g. calculus.

To prevent gum disease, he recommended cleaning the teeth, massaging the gingiva with an astringent liquid, and washing the mouth with wine and water. Because of the spongy appearance of the gingiva that had similarity to gingiva he observed in patients with scurvy, Fauchard called the phenotype scurvy of the gums. Fifty years later, the Scottish surgeon and scientist John Hunter (1728–1793) published A Practical Treatise on the Diseases of the Teeth intended as a supplement to the Natural History of those Parts, his second and less popular book on teeth and tooth related structures. Hunter postulated that gradual loss of alveolar bone, associated pocket formation and gingival recession would inevitably result in tooth loss. He considered the process to be disease when it occurred early in life, but the result of natural aging in the elderly.

Among gingival diseases, Hunter distinguished between cases that

resembled Fauchard s scurvy of the gums and cases of overgrown fibrotic tissue [15]. Achievements in microbiology, a novel scientific discipline in the 19th century, changed the way periodontal diseases were viewed. Two German physicians, Robert Ficinus (1809–1852) and Adolph Witzel (1847–1906), deserve credit for associating bacteria with periodontal tooth loss. In Europe, the term alveolar pyorrhea was coined to describe any form of periodontal disorder unrelated to the aging process. According to Witzel, in patients exhibiting alveolar pyorrhea, the gingiva forms a pocket that allows bacteria to infect and destroy the underlying periosteum and ultimately alveolar bone.

In the USA, John M. Riggs (1810–1885), also known as the father of periodontics, treated diseased pockets by painstakingly thorough calculus removal, curettage of soft tissues, and implementation of meticulous individual oral hygiene. He was convinced that all stages of periodontal disease, from the earliest signs of inflammation to tooth loss, were attributable to the same local etiology, i.e. calculus.

Reportedly, Riggs rigorous procedure healed more than 90% of his patients, a huge improvement over any previously known treatment. His success was soon recognized. In 1869, the Connecticut Valley Dental Association passed a resolution acknowledging Riggs as the first person ever to treat gum inflammation successfully [16]. At the 1877 meeting of the American Dental Association, the Germany-born physician Frederick H. Rehwinkel (1825–1889) presented a paper on pyorrhea alveolaris, and ever since has been credited for introducing the European term to the American dental literature [17]. The term pyorrhea alveolaris was quickly adopted by the dental community. It persisted deep into the 20th century, despite being rather a poor choice for the disease it purported to describe.

Focal infection theory

Willoughby D. Miller (1853–1907), whose work was very much influenced by Robert Koch, is best known for his groundbreaking ideas on the etiology of caries. He also postulated a role for bacteria in the etiology of alveolar pyorrhea [18], and concluded that, in the presence of predisposing factors, many bacteria found normally in the mouth can cause periodontal disease (e.g., non-specific plaque hypothesis). Miller advocated that such bacteria could also play a role in the etiology of many other diseases in humans. He coined the expression focus of infection [19], but stopped short of promoting eradication of infected teeth to prevent or treat systemic illnesses. This step was made by Frank Billings (1854–1932), a highly respected American physician and academic leader. Billings and his student and colleague Edward C. Rosenow (1875–1966) promoted the theory of focal infection in the USA [20,21].

The focal infection paradigm was quickly adopted by many dentists and physicians, especially surgeons in the USA. Its clinical implementation, which was further promoted by substantial improvements in asepsis, led to uncountable unwarranted tooth extractions, tonsillectomies and other surgical procedures [22]. In 1928, Holman publicly questioned the validity of the focal infection theory. Its importance started to decline in the 1930s as evidence accumulated indicating that surgical removal of suspected foci has no beneficial effect on the medical status of affected patients [23,24].

Epidemiology

General trends Gingival and periodontal diseases occur globally and

among virtually all populations that have been studied to date [25]. Clinical signs of periodontal destruction may be absent in individuals of any age, but there is little evidence supporting the existence of periodontitis-resistant populations. However, two defined apparently resistant cohorts with no access to dental care were described in Namaqualand and Crossroads (South Africa) [26,27].

The World Health Organization performed extensive surveys of the periodontal status of potentially under-served populations around the globe, especially in developing countries [28]. Using the Community Periodontal Index for Treatment Needs to assess prevalence, a majority of subjects examined had gingivitis and 10–15% of adults had periodontal pockets ‡6 mm deep. In contrast to common belief, periodontal diseases are not the most important factor for tooth loss in many Asian and most African populations. In fact, surveys have indicated that, in Africa, most people retain the majority of their teeth throught their lives [29,30].

In the USA, based on estimates obtained from the National Health and Nutrition Examinations Surveys performed from 1988 to 1994 (NHANES III) [31], the overall prevalence of moderate to severe signs of periodontitis among adults was 7.3%, corresponding to one affected adult person in [20, 25, 32]. Approximately one in five adults had slight periodontitis, one in ten had moderate periodontitis, and one in 30 showed signs of severe periodontitis. The prevalence of periodontitis increased with population age, but the prevalence of more severe forms peaked at 70 years and leveled off thereafter [25]. Periodontitis was more frequently diagnosed in men than in women. Substantial racial and ethnical disparities were observed. Surveys performed in Europe have corroborated the suspected secular trend of prevalence reduction in periodontal diseases among adults [33,34]

A comparison over a 30-year period of the prevalence of various clinical signs of periodontal disease in Swedish subjects aged 20–70 years revealed remarkable changes (169). [35]. Over the surveyed period, the proportion of periodontally healthy individuals improved from 8% to 44%. The increment compensated for the decrease in the proportion of individuals with gingivitis or moderate alveolar bone loss. In contrast, the proportion of subjects exhibiting signs of severe periodontal disease was small and did not change over time. Improvements in oral hygiene, changes in life style and adoption of less risky behavior, in particular recognition of deleterious smoking effects, have been proposed to explain the unexpected decline.

Aggressive forms of periodontitis are defined by rapid localized or generalized loss of the supportive periodontal structures, and occur in family clusters in otherwise medically healthy subjects [36]. Aggressive forms can affect the primary or permanent dentition. Typically, susceptible patients are less than 30 years old at disease onset[37]. The similar phenotypes of aggressive periodontal disease are probably the clinical expression of multiple disease forms with discrete etiologies.

A review concluded that aggressive forms of periodontitis have a low prevalence in most regions of the world, occurring in 0.1– 1.0% of the population [38]. For reasons that are unclear at present, this disease form is seen substantially more frequently among young black subjects. A prevalence of 2.6% was reported in a sample representative of high school students in the USA [39]. Even higher disease prevalences of 6.5% and 7.6% were observed in two cohorts of adolescents and

young adults in Uganda [40] and Morocco [41], respectively.

These subjects were carriers of *A. actinomycetemcom*itans clone JP2, which is endemic in Morocco. Carriers of the JP2 clone, who were free of clinical disease signs at the first examination, had a relative risk of 18.0 CI 7.8–41.2) of experiencing periodontal attachment loss during the 5-year observation period [42].

Although earlier reports by Saxen [43] showed a female majority among subjects with early-onset aggressive periodontitis, a more recent national survey conducted in the USA did not corroborate this observation [44]. Furthermore, based on the results of a genetic segregation analysis performed in 100 families in the USA, the aggressive disease trait has an autosomal dominant inheritance pattern [45]. This contrasts with the autosomal recessive inheritance pattern identified in northern Europe[46], suggesting a different pathway to disease for each of the two populations.

Necrotizing periodontal diseases feature ulceration and necrosis of the interdental papillae, spontaneous bleeding, pain and a removable pseudomembrane [47]. Advanced cases involve alveolar bone resorption, may be generalized, and may lead to fever, malaise and lymphadenopathy [48].

Necrotizing periodontal diseases are observed frequently in parts of Africa, Asia and Latin America, mainly in socially disadvantaged children. For example, in an urban South African clinic population, the prevalence of acute necrotizing ulcerative gingivitis was 3%. Males were more frequently affected than females. Seventy-three per cent of the patients were children between 5 and 12 years old. The seasonal occurrence varied greatly, with most cases diagnosed in the summer (relative risk 6.57; 95% CI 4.96–8.70) [49].

In contrast, necrotizing periodontal disease is now rarely observed in the general population of Europe and North America, as confirmed in a recent study among 18- to 22-yearold military recruits in Switzerland [30]. A high prevalence of necrotizing periodontal disease has been observed in HIV-infected subjects [50 - 52]. Based on this finding, the presence of necrotizing periodontal disease has been recommended for use as a surrogate marker of HIV-associated immune deficiency and AIDS (125). [53].

Periodontal disease classifications. Times of transition and consolidation.

At the dawn of the 20th century, the realization that alveolar pyorrhea can be treated led to recognition of periodontia as a dental specialty. The professional organization of periodontists now known as the American Academy of Periodontology was established in 1914 as the American Academy of Oral Prophylaxis and Periodontology. In Germany, the Arbeitsgeme inschaft fu¨r Paradentosen Forschung was formed in 1924 with the goal of establishing an open communication platform for academicians and practitioners.

However, the much needed information exchange was impeded by decidedly inconsistent terminology. Over subsequent decades, periodontists on both sides of the Atlantic met repeatedly to develop countless classification systems that reflected scientific progress as well as clinical utility. As a result of this effort, new nomenclatures were published at arbitrary intervals by professional bodies such as the American Academy of Periodontology, the American Dental Association, the Arbeitsgemeinschaft fur Paradentosen Forschung and the World Dental Federation, among others. In addition, classifications were also contributed by individual authors.

One of the major biomedical accomplishments of the 20th century was the recognition that formal hypotheses can be tested in the clinic. Application of

quantitative methods to clinical problem solving, albeit implemented slowly in most dental disciplines, had profound effects on the classification of periodontal diseases. The paucity of scientific evidence in support of periodontosis and occlusal trauma as classes of periodontal disease was initially acknowledged at the 1966 World Workshop in Periodontics [54], and formalized 11 years later at the International Conference on Biology of Periodontal Disease. Only two classes of periodontal disease remained – juvenile periodontitis and chronic marginal periodontitis [55] – and these constituted the 1977 American Academy of Periodontology classification system.

In the meantime, the studies on experimental gingivitis by Harald Loe and his collaborators at the Royal Dental College in Aarhus, Denmark, ushered in the plaque era of periodontology. Using novel index systems to assess plaque and gingivitis [56,57], they provided unequivocal experimental evidence for a direct relationship between the presence of dental bacterial plaque and gingivitis [6, 58, 59]. In addition, they demonstrated the full reversibility of all clinical signs of gingival pathology when oral hygiene was re-established. There is no question that the nature and results of these ground-breaking studies had a profound effect on most aspects of clinical periodontology. Moreover, a destructive form of periodontal disease, most frequently observed around the central incisors and first molars in young people, initiated renewed interest in the disease class previously known as periodontosis. Baer [60] and Manson & Lehner [61] published initial clinical reports that were followed by studies of host defense mechanisms[62-64]

Because it was frequently diagnosed in adolescent patients, the form was re-named juvenile periodontitis. At the time of the 1977 International Conference on Biology of Periodontal Disease, the majority opinion among dentists was that, without treatment, gingivitis progresses to periodontitis, at a relatively constant rate, ultimately resulting in tooth loss. This view was challenged when Loe et al. [65] presented their results on the natural history of periodontal diseases. In most subjects, the presence of plaque and gingivitis did not lead to severe periodontitis or tooth loss.

These and other findings were reflected in a new classification that was first suggested at the 1986 World Workshop in Clinical Periodontics and modified at the World Workshop in Clinical Periodontics in 1989 [66]. The revised system distinguished five classes of periodontal disease. In comparison to the 1986 taxonomy, a new class was introduced for periodontal diseases linked to systemic conditions.

Finally, in 1993, the European Academy of Periodontology (now the European Federation of Periodontology) was founded at the 1st European Workshop on Periodontology. The European Academy of Periodontology adopted the American Academy of Periodontologys 1989 classification but suggested that an improved system should be considered based on three major classes: early-onset periodontitis, adult periodontitis and necrotizing periodontitis. Each of these was further defined by secondary descriptors (distribution within dentition, progression rate, treatment response, relation to systemic disease, microbiological characteristics, etc.) [67].

1999 International Workshop for Classification of Periodontal Diseases and Conditions

The classification (2010) was implemented based on recommendations by the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions [68]

In addition to increasing the number of disease classes from five to eight, the

revision included several substantial deviations from preceding classifications.

Classification limitations and next steps

Periodontal disease classification has evolved over a long time period. The purpose of disease classification is to unambiguously link clinical phenotypes, defined by clinical observations and simple laboratory investigations, with diagnoses and ultimately disease-specific therapy . The currently used system is a combination of broadly defined classification elements such as location, etiology and pathology. Examples have been described in the literature [69,70], and concern non-inflammatory destructive periodontal disease or atrophy. A weakness of many disease classifications is substantial overlap among classes. An example is the definition of aggressive periodontitis (class III) as rapid attachment loss and bone destruction.

To mitigate the risk of mis-classification, selected biochemical (e.g. interleukin-1b and prostaglandin E2) and microbiological (e.g. *Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans*) secondary identifiers have been proposed [36]. Another concern is the sensitivity and specificity of the methods used to collect patient information.

Data on change in probing depth, clinical attachment and radiographic bone level are collected as the result of disease history. The fundamental mechanisms involved in onset and progression of most diseases, including those affecting the periodontium, remains incomplete. The already substantial amount of knowledge acquired from clinical and laboratory research regarding the etiology and pathomechanisms responsible for periodontal diseases has not yet paid off in terms of development of potent preventive and therapeutic measures for the individual patient. In the not so distant future, such information could be used for identification of phenotypic differences among patients suffering from the same disease, subsequently leading to individualized, patient-specific therapies [71]

The new periodontal classification

In the last 30 years, the periodontitis classification has been repeatedly modified in an attempt to have a result of emerging science. The 2017 Workshop agreed that, according to current knowledge of pathophysiology, three forms of periodontitis can be identified: necrotizing periodontitis [72], periodontitis as a manifestation of systemic diseases [73] and forms of disease previously recognized as "chronic" or "*aggressive*", now grouped under one category, "*periodontitis*" [74].

The 2017 World Workshop Classification system for periodontal and peri-implant diseases and conditions was developed in order to accommodate advances in knowledge derived from both biological and clinical research, that have emerged since the 1999 International Classification of Periodontal Diseases. [75] The result is a redesigned disease classification framework that guides comprehensive treatment planning and allows for a personalized approach to patient care.[76]. Highlights from the updated classification include a multi-dimensional staging and grading system for periodontitis classification, a recategorization of various forms of periodontitis, and the inaugural classification for peri-implant diseases and conditions. Of extreme importance, the new classification includes clinical health for the first time, and distinguishes an intact and a reduced periodontium throughout. Also, the term *'aggressive periodontitis'* was removed, creating a staging and grading system for periodontitis that is based primarily upon attachment and bone loss and classifies the disease into four stages based on severity

(I, II, III or IV) and three grades based on disease susceptibility (A, B or C).

The new periodontal classification system allows clinicians to better categorize patients' oral health based on clinical and radiographic findings. Now that the oral-systemic link is part of the classification system, it will help patients become more involved in knowing the state of their diseases [76,77]. Talking to patients about their classification can lead to better treatment acceptance and understanding. The new classification is complex and will take time to fully incorporate into patient care globally [77].

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