

## SOME MODERN ASPECTS OF PERIODONTAL DISEASE

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خلال السنوات الثلاث الأخيرة تغيرت الآراء وبشكل كبير حول الأسباب والآلية المرضية وكذلك طرق معالجة الأمراض اللثوية. ففي هذه الفترة جرى تقويم للأسباب المساعدة لهذه الأمراض، وأجريت تجارب عملية دقيقة ودراسات بيئية لتلك المشكلة. ولم تعد معالجة أمراض اللثة فن كما كانت عليه الحال مع نهاية القرن التاسع عشر، بل أصبحت علماً يعتمد على البحث العلمي.

فالتقيح السني السنخي البيوريا كان يعتبر مرضاً متعدد الأسباب ذو انذار سيء مماثل لنخر الأسنان حيث يسبب فقداً للأسنان في المجتمعات المختلفة وقد افترض أنه مرض يصيب المتقدمين في السن أو أولئك المصابين بمرض سنية بسبب سوء الأطقم أو أمراض عامة أو صحة سيئة.

إن اكتشاف العلاقة بين اللويحات المخاطية والتهاب اللثة وكذلك اختلاف تركيب الجراثيم الموجودة تحت اللثة من مكان إلى آخر في الحفرة القموية ومن شخص إلى آخر خلقت اقتراحات وأساليب جديدة لمعالجة الأمراض اللثوية المختلفة.

إن الهدف من هذه المقالة هو إعطاء ملخص لبعض الخصائص الحديثة لأمراض اللثة.

During the last three to four decades, extensive changes in opinion concerning the etiology, pathogenesis, and treatment of periodontal disease have taken place. During these decades, contributing factors are standardized and controlled trials, as well as epidemiological studies, were performed. Periodontics is no longer an art as it was at the end of the 19th century- it is a science based on research.

Pyorrhea alveolaris or periodontitis has been considered a multifactorial disease with bad prognosis which, together with caries, causes loss of teeth in the population. It was supposed to affect most of the population with age progress, trauma from occlusion, systemic diseases, and bad oral hygiene.

The discovery that plaque was the cause of gingivitis, and that the subgingival microflora differed in composition between sites, teeth, and individuals created new suggestions and demands for the treatment of periodontal disease. The aim of this paper is to summarize some modern aspects on periodontal disease.

### Epidemiology

In 1944, Allen<sup>1</sup> found that periodontal disease, as a cause of tooth extraction, was limited to 13% of the consecutive registrants at the University of Michigan Dental School Clinic, indicating a low prevalence of severe of periodontal disease. The development of periodontal and oral hygiene indices in the 1950s were aimed to ascertain periodontal status of a population.<sup>24</sup>

Studies based on these indices indicated periodontal disease to affect virtually all individuals worldwide. Page and Schroeder<sup>5</sup> however, questioned the high prevalence of periodontal disease, the progression from gingivitis to periodontitis, and periodontitis as a single disease. Recent studies<sup>6-10</sup> show that both the severity of periodontal disease and oral hygiene vary considerably and that a relatively small proportion of individuals within a population account for most of the observed periodontal breakdown and tooth loss.<sup>11,8,1</sup>

Epidemiological data<sup>12-14,10</sup> do not deny that gingivitis and periodontitis are related, "that gingivitis does not always progress to initiate periodontitis, gingivitis does precede periodontitis, and once periodontitis is initiated, gingivitis is not

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necessary for the destruction of the deeper periodontal structures to continue".<sup>15</sup>

The introduction of severity indices in the 80s revealed that 8 to 15% of the adult population suffered from severe periodontitis, which when untreated causes tooth loss.<sup>16</sup>

Even from developing countries, reports pointed out that the prevalence of severe periodontal disease is limited.<sup>17,10</sup> These facts explain why periodontitis is no longer looked upon as a cause of extensive tooth loss after 35 years of age,<sup>18,19</sup> as was suggested during the 50s and 60s.<sup>20-22</sup> After 50 years of age, more teeth are lost because of periodontitis than of caries but still periodontitis does not count for 50% of the tooth loss.<sup>23</sup>

However, most of the present national epidemiological data on the prevalence of severe periodontitis "is notorious for its low reliability". As a basis for making any conclusion about prevalence and trends in advanced periodontitis and extent of periodontal diseases, further investigations and analyses must be performed.<sup>24</sup>

### Gingivitis and Periodontitis

The prevalence of gingivitis is virtually 100% in a population with no oral hygiene and declines with improved oral hygiene.<sup>25</sup> The inflammatory changes in the gingiva are related to the colonization of the tooth surface in the gingival crevice.<sup>26</sup> This marginal plaque consists of unspecific bacterial flora.<sup>27</sup>

Several studies have shown that gingivitis can stay for years without progressing into periodontitis.<sup>12,28,29</sup> However, this does not mean that gingivitis should be left untreated.

Periodontitis is usually classified into initial, moderate, and severe, or chronic and rapid progressing periodontitis, depending on the amount of bone loss or speed of progression, respectively. Localized periodontitis affecting few sites, or teeth, is more common than the generalized one, which is independent of the oral hygiene status, severity of gingivitis, and accessibility to professional treatment.<sup>30-32,10,25,33</sup> However, in populations where the oral hygiene is good, severe periodontitis seems to be less frequent even if it is present.<sup>11,34,35,36</sup>

The prevalence of periodontitis has been elucidated by the World Health Organization presenting data on periodontal condition from many countries in its Global Oral Data Bank using the

CPITN criteria.<sup>37</sup> Of the 46 surveys from 33 countries listed by Pilot and Barmes,<sup>38</sup> 39 surveys show pockets equal or more than 6 mm in 0 to 19% of the dentated population aged 35-44 years. Five of the remaining surveys show severe periodontal disease in 28-34% of the same age group. These data indicate that periodontal disease may not be as large a public health problem as has been believed until now. In the National Survey of Oral Health in the United States in 1985-1986 on employed adults and seniors,<sup>39</sup> 80% of the males and 73% of the females, 18-64 years of age, had at least one site with loss of periodontal attachment; amongst the seniors, 65-80 years of age, the figures were 98% and 94%, respectively; 4% of the 18-64 year-olds and 41 % of the 65-80 year-olds were edentulous.

The figures are in agreement with earlier report but it also show that there is a need for a lot of periodontal treatments in the population, besides those affected by severe periodontitis.

Reports from different places around the world show a prevalence of severe periodontitis in around 8-10% of the population; Sweden 8%; England 7%; The Netherlands 10%; Italy 9.6%; and Sri Lanka 8%.<sup>35,40-42,14</sup>

The acute necrotizing ulcerative gingivitis (ANUG), which after World War II was a rather frequent disease, in 1-2% among recruits,<sup>43</sup> is today an uncommon disease in the industrialized countries. However, it is a common disorder in connection with AIDS.<sup>44</sup>

Modern literature not only distinguishes between gingivitis and periodontitis but also includes classification of periodontitis into:

1. *Adult periodontitis* is a slowly progressing periodontitis of adults.
2. *Rapidly progressive periodontitis*, with a rapid clinical cause in young adults and adults, sometimes classified as "post-juvenile periodontitis".
3. *Localized juvenile periodontitis* with onset of puberty. It is a rapidly progressive and localized periodontitis which is hereditary (autosomal recessive).
4. *Pre-pubertal periodontitis* is an extremely rare and rapidly progressive periodontitis of the deciduous dentition affecting all teeth (generalized type) or individual teeth (localized type).<sup>45,46,47</sup>

The classification of periodontitis is based on the speed of progression and onset in various age groups. The discovery that periodontally affected persons, teeth, or root surfaces, show different combinations of bacteria is not a motivation of using different diagnoses of periodontitis but is an important knowledge when treating the infection (antibiotics or not).

Periodontitis is often localized. The microbiology of these sites vary, and it is too complicated to evaluate the microbiology to satisfy the clinician. The future will probably explain the view of local acting bacteria. There is a need for more research of a host-parasite relationship to solve this question.

### Periodontal Disease and Age

Epidemiological studies<sup>48</sup> reveal that attachment loss increases significantly with age. At the same time, it is reported<sup>49</sup> that periodontal disease is related to the oral hygiene status, and that the effect of age on the progression of periodontitis could be "considered negligible when good oral hygiene is maintained". Burt et al<sup>50</sup> reported that only 8% of the Americans under 63 years of age suffered from severe periodontitis (attachment loss  $\geq 6$  mm), but that figure was 34% for patients over 65 years of age. Even if this difference is real, more attention has been focused on the relationship between periodontal disease and oral hygiene than on age.<sup>50</sup>

The increase in periodontitis by age may depend on increasing plaque accumulation and long time exposure.<sup>51</sup> Africa et al<sup>52</sup> and Reddy et al<sup>53</sup> observed that unsatisfactory oral hygiene, corresponding to high plaque levels, does not always result in destructive periodontal disease. Therefore, the increasing periodontal bone loss by age can also be a result of reduced plaque tolerance in the elderly.<sup>31</sup> In a review article, Van der Velden<sup>53</sup> concludes that periodontal tissue in elderly person is more susceptible to periodontal disease and overshadows the time of plaque exposure.

In a 10-year longitudinal study on 82 subjects with untreated periodontal disease using the PI index,<sup>2</sup> Buckley and Crowley<sup>54</sup> found that progression was slow in all age groups for teeth initially free of periodontal disease and with mild gingivitis. Subjects more than 35 years of age with severe gingivitis showed more rapid bone destruction than

younger persons. In contrast, if destructive periodontal disease develops in younger people, this will then show a poorer prognosis.

Pilot and Schaub<sup>55</sup> noticed that not all teeth are equally affected by periodontal disease. The upper first and second molars, and the lower front teeth have been found to be the most frequently affected.<sup>31,30</sup> In juvenile periodontitis, the periodontal breakdown is, however, homologous usually affecting the incisors and the first molars in both jaws.<sup>56,57</sup>

The number of teeth or sites affected by periodontal disease vary from patient to patient.<sup>58</sup> Most patients experience destructive periodontitis at relatively few sites<sup>59</sup> and only a few show generalized periodontal bone loss.<sup>58</sup>

Explaining factors to the different patterns in rapid progressing periodontal diseases can be the pathogenicity of bacteria, the immune response and the patients' age when infected, as well as its genetic factors.

However, "other factors that may affect or enhance the accumulation and growth of plaque, or preclude, or interfere with its removal, and local and systemic or constitutional factors that may alter resistance or susceptibility of the tissues of the periodontium to bacteria and other noxious substances, are also of importance".<sup>60</sup>

### Geographic and Genetic Factors

Comparison between Norwegian academicians and teaplant workers in Sri Lanka of the same age revealed a higher prevalence and severity of periodontitis among the teaplant workers. The latter are poor and present a bad oral hygiene, which in itself can explain the difference between the groups but a reduced resistance against bacterial plaque as an explaining factor has also been assumed.<sup>6,14</sup>

The spread and degree of severity of certain periodontal diseases seem to follow geographic and ethnic lines. A possible genetic association to the prevalence of severe periodontitis has also been pointed out when comparing the frequency of severe periodontal disease in Tonga and in Western Samoa. The populations in these islands have strictly lived separately, most obviously been on similar diet, maintained a similar oral hygiene and are of similar ethnic origin,<sup>8</sup> but still the inhabitants of Tonga show a more advanced periodontitis. Attempts about genetics in periodontitis have been made by Cockey et al<sup>61</sup> and by Michalowicz.<sup>62</sup>

The prevalence of juvenile periodontitis varies from 0.8% among blacks, 0.2% in Asians, and 0.02% among whites.<sup>63,25</sup> In Finland, the prevalence has been reported to be 0.1 % according to Saxen.<sup>64</sup> However, the prevalence of juvenile periodontitis has not been fully investigated worldwide mainly because of diagnostic problems. The great variance in prevalence has been suggested to depend on genetic factors.<sup>65</sup> Newman<sup>66</sup> suggested a familiar pattern resulting from: 1) genetic predispositions to specific groups of bacteria; 2) a genetically determined immuno-deficiency; and 3) faulty or impaired formation and maintenance of periodontal tissue integrity.

In a recent research on juvenile periodontitis,<sup>67</sup> the hereditary basis has been documented. Systemic diseases as genetic disorders (Papillon-Lefevre syndrome, acatalasaemia, Down's syndrome, Chediak-Higashi's syndrome), blood disorders (neutropenia, leukemia), hormonal diseases (diabetes mellitus), and contagious diseases (AIDS) have been proven to be associated with rapid progressive periodontal disease.<sup>68,44</sup>

#### **Bacteriology in Gingivitis and Periodontitis**

Bacterial plaque has long been believed to constitute the primary extrinsic etiologic factor for inflammatory periodontal disorders.<sup>69,26,70-73</sup> The recently developed bacteriological techniques have permitted cultivation of most microorganisms in the oral cavity.

The number of microbial species recorded in the oral cavity is steadily increasing and, presently, over 400 species have been described.<sup>74</sup> However, many bacterial strains isolated from the oral cavity remain to be classified. Analysis of the microflora of the gingival crevice showed the different compositions in cases of healthy periodontium, gingivitis, and periodontitis.<sup>5,47,71,74-77</sup> These discoveries may enable researchers to reanalyze the concept of specific bacteria in periodontal disease against the non-specific bacterial hypothesis.<sup>78</sup>

The bacteriology, associated with gingivitis, has not been characterized in the same extensive way as the bacterial flora of the healthy periodontium and the periodontitis.<sup>79</sup> Microscopic studies in experimental gingivitis in man<sup>27</sup> showed three phases in the changes of the microbiota during a two-week period. In the first phase, gram-positive bacteria dominated the microbiota; while in the

second phase, filamentous organism appeared and during the third phase, spirals and spirochetes characterized the bacterial plaque. Among the identified anaerobic bacteria, fusobacterium nucleatum and bacteroides intermedius were common findings.<sup>58</sup>

In the early stages of periodontitis, the bacterial flora is similar to that of gingivitis<sup>80,81</sup> but in the more advanced cases of periodontitis there is a predominance of gram-negative anaerobes as bacteroides gingivalis. In juvenile periodontitis, attention has been paid to the presence of actinobacillus actinomycetecomitans as well as to fusobacterium nucleatum. Leukotoxin produced by actinobacillus actinomycetecomitans has the capacity to interfere with cell vitality and function.<sup>82</sup>

Whether specific microbes can develop periodontal disease is still unknown. Conclusively, we still have to rely on the unspecific bacterial plaque flora theory until the significance of specific bacteria may be verified.

#### **immunology**

The loss of attachment level is associated with the patient's reaction to bacteria. In gingivitis, the connective tissue is infiltrated with immunocomplement cells like T- and B- lymphocytes, plasma cells, and monocytes. With increasing long standing inflammation, the supporting fiber components and the ground substance of the gingiva are degraded and the gingiva is gradually converted into granulation tissue. It is well known that there is a local antibody formation in the gingiva, and an increased antibody level in the gingival fluid. Antibodies against oral bacteria, are also recognized in the peripheral blood.<sup>83</sup> Bacteria on the gingiva are able to activate immuneresponse, which, depending on degree of reaction, will result in protection or destruction of the periodontal tissues. In recent years, special methods have been developed to quantitate the amount of formed antibodies so as to determine the antibody titer. Tolo et al<sup>84</sup> investigated the humoral immune-response against eight isolated strains of bacteria from pathological pockets of patients with periodontitis. They found that bacteroides gingivalis gave a high increase of IgG and IgA antibodies in serum. The serum concentration of antibodies against bacteria from the gingiva in adult patients without periodontitis is interpreted as a response of protection against oral bacteria.

Increased serum concentrations of antibodies against specific bacteria have been found five times higher from patients with periodontitis than from periodontally healthy patients.<sup>85</sup> Concerning the specificity of antibodies, most of the antibodies are unspecific, only a few of the locally produced antibodies are specific to known periodontal pathogens. The significance of the antibody titer is not quite clear. Conclusively, periodontitis is a multicausal disease, but still investigations are needed to prove if there are cases of periodontal disease caused by specific bacteria.<sup>86,87</sup>

High antibody titers might be indicators of risk patients, who may be affected by rapid progressive periodontal disease when the defense mechanism is weak.

### Burst Theory

Recently, the progression of periodontal disease has been facing a new theory — the Burst Theory — introduced by the Forsythe group in 1984.<sup>88</sup> The opinion that chronic periodontitis is a slow, continuous progressive disease has been questioned. The Forsythe group suggests that periodontitis progresses in intervals with breakdown followed by repair and long period of low or non-inflammatory activity. The active phase is supposed to continue for hours to several days, if the reparative phase in total is less than the destructive phase, the damage of the periodontal tissues will accumulate over time and age. Longitudinal observations from one year up to six years of patients with destructive periodontal disease indicate that certain sites significantly lose more attachment level in a short time than other sites. In the long run, some of these did not show any attachment level loss at all or even obtained gain of attachment.<sup>89-93,59</sup>

The Burst Theory is in agreement with the progression of certain systemic diseases. Rheumatoid arthritis is an example of such a disease with periodic exacerbations. The data presented by the Forsythe group are based on limited investigations and must be verified by others being accepted.<sup>94-96</sup> Furthermore, there is a need for a simple disease activity index to evaluate when the disease is active and treatment is needed. Consequently, there are at present no possibilities to identify patients *in risk* for developing destructive periodontal disease.

### Trauma from Occlusion

In 1901, Karolyn<sup>97</sup> indicated that trauma from occlusion has been connected with periodontal disease. Interfering cusps have been ground and biteguards were constructed to perform harmonic occlusion. In the beginning of the 60s, Waerhug<sup>98</sup> strongly suggested that trauma from occlusion was not an etiologic factor causing periodontal disease. Various investigations<sup>99,100</sup> in dogs and squirrel monkeys support Waerhug. Today, it is believed that trauma from occlusion can not initiate periodontal disease, but may aggravate an existing one.<sup>101</sup> Trauma from occlusion needs, however, further investigation to elucidate its role in the progression of periodontal disease.

### Treatment Needs

It is important to distinguish between the periodontal treatment need on the individual and the community level. The PTNS,<sup>102</sup> and CPITN,<sup>37</sup> indices were introduced to estimate type, severity, and resources needed for periodontal treatment in the community. The CPITN index has, however, been criticized as it is unsatisfactory "for the study of prevalence, severity, and extent of periodontal diseases" and that it underestimates the severity of periodontal disease in a given population.<sup>103</sup> The prevalence of severe periodontitis of 8-15% using the CPITN index has been misinterpreted believing in a low periodontal treatment need. The fact is that the population is also suffering from moderate and initial periodontitis as well as from gingivitis, supra- and subgingival calculus. As there are no possibilities to identify patients in risk for developing destructive periodontal diseases, the dental profession has to treat all kinds of periodontal diseases, including gingivitis. It would be unethical, as in general medicine, not to treat the non-fatal diseases. The quality of life and elimination of disease are, after all, the main concerns in all health care.<sup>104</sup>

### Treatment

The treatment of periodontal diseases focus on removal of supra- and subgingival debris. Recent investigations have, however, shown that the healing of periodontal disease is similar, if subgingival scaling and root planing is performed with hand, sonic, ultrasonic instruments or special diamonds,<sup>105,106</sup> with or without surgery,<sup>91,107-109</sup> and

if regular oral hygiene and maintenance care are performed. It has also been shown that supragingival plaque removal without subgingival instrumentation is insufficient to arrest progression of manifest periodontitis.<sup>110</sup>

In cases of persisting pathologic pockets and limited access to subgingival concretions, periodontal surgery is indicated. However, in shallow pockets it has been shown that there is loss of attachment but in deep pockets reduction, when periodontal surgery is performed.<sup>107,109</sup> However, "scaling and root planing alone (non-surgery group) required more than twice the number of hours for active treatment than scaling and root planing in conjunction with surgery (surgery group)".<sup>107</sup> This gain in time using periodontal surgery in deep pockets is of great cost-benefit importance.

In the 1960s, reconstructive surgery was performed with bone implants. The results were disappointing as new attachment was rarely obtained. Today new attachment can be obtained by "guided tissue regeneration surgery", where various inserted materials, as expanded teflon and resorbable materials (PGA, Vicryl, collagen), have been used in well controlled animal and human trials.<sup>111-114</sup> However, this surgery must still be looked upon as experimental surgery and need further investigations before being used by the general practitioner.

In mucogingival surgery where the aim is to increase the width of the attached gingiva, the indications on community basis are limited.<sup>115</sup> However, on individual basis, mucogingival surgery is indicated for aesthetic, phonetic, orthodontic, postsurgical, periodontal, and prosthetic reasons. The explanation is that attached gingiva does not seem to be as important for the maintenance of a healthy periodontium as it has been claimed before.<sup>116</sup>

Refractory cases are cases of periodontal diseases, which show progression in spite of meticulous mechanical removal of bacterial plaque and subgingival calculus. They have been connected with systemic diseases. The treatment of such cases, if existing, has been regular oral hygiene, broadspectrum antibiotics, and intense maintenance care. Antibiotics should, however, be used with restriction and should only be administered when necessary, i.e., when the patient needs them

for systemic reasons and in refractory cases where specific bacteria have been identified and tested.

### Maintenance Care

The immediate maintenance care after periodontal surgery, has been shown to be of greatest importance for the long term result. Westfeld et al<sup>117</sup> showed that healing will improve during the first three postoperative months, if postoperative check-ups were performed each 2-4 weeks compared to each 12 weeks. The length of the intervals of the maintenance care can be made each 3, 6, or 12 months depending on the patients cooperation and skill.<sup>104</sup>

Invention of a disease activity index will, in the future, not only be of importance for where and when to treat periodontal diseases, but also for determining the intervals for the maintenance care.

### Conclusions

The prevalence of severe periodontal disease, which when untreated will result in tooth loss, seems to be limited to about 10% of the population.

Mainly, periodontal disease does not affect all teeth or surfaces in a similar way. The bacterial flora is different on various sites on the same tooth, and may vary between teeth in the same mouth as well as between individuals.

Bacteria, as the only etiological factor to periodontal disease, should regularly be removed for the prevention and treatment of periodontal disease. The importance of specific bacteria in the etiology of periodontal disease is still under discussion. The progression of periodontal disease is affected by the condition of the organism.

Immunological factors seem to play an important role. Increased antibody titer against bacteria may be of importance but its role is still not clear. The importance of genetics in the development of periodontal disease needs further clarification.

Periodontal disease, including gingivitis, has to be treated to fulfill the ethics of the dental profession. Its treatment includes supra- and subgingival plaque removal. However, surgical or non-surgical subgingival plaque removal with hand, sonic, ultrasonic instruments, or diamond burrs gave similar results.

Maintenance care after surgery is of greatest importance for success. However, the use of anti-biotics in the treatment of periodontal disease should be restricted.

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