Public Health Implications of Recent Research in Periodontal Diseases

Brian A. Burt, BDS, MPH, PhD

Professor and Director Program in Dental Public Health School of Public Health The University of Michigan Ann Arbor, MI 48109-2029

Abstract_

Knowledge of the epidemiology, natural history, and bacterial etiology of the periodontal diseases has advanced considerably as a result of research conducted through the 1980s. Prevention and control of these conditions, however, remains mechanical, cumbersome, and often impractical, based as it is on bacterially nonspecific plaque removal for an indeterminate period. This research has not yet changed the content of public health programs, but it does affect the way the programs are applied. Because severe, generalized disease seems to be less prevalent than previously thought, the need of regular, routine professional care for everybody is questioned. Professional care in a public health context is likely to be more efficient when targeted toward those with severe disease. Dental health education for personal oral hygiene is still supported by scientific studies, though a targeted approach and careful assessment of educational content is needed. Until predictive screening methods for identifying susceptible individuals are developed, selection of priority groups for education and treatment should be guided by epidemiologic data.

Key Words: periodontitis, gingivitis, public health, dental health education, professional care, epidemiology

Introduction .

Humankind has been ravaged by disease epidemics since the beginning of recorded history (1-3). Until recent times, there was little people could do about them other than pray; indeed, the public health response to the British cholera epidemic of 1831 was an official prayer for deliverance (4). Until about the mid-1950s, "pyorrhea" was like cholera in the early 19th century: one got it or one did not; but either way, one could do little about it. When contracted, "pyorrhea" led to its own kind of mortality—total tooth loss.

Systematic study of the periodontal diseases came late (5-8). Early epidemiologic research presented a pic-

Paper given at the symposium "Periodontal disease: is it a public health problem?," at the annual meeting of the American Public Health Association, New Orleans, LA, October 18-22,1987. Send correspondence and reprint requests to Dr. Burt. Manuscript received: 11/18/87; returned to author for revision: 1/22/88; accepted for publication: 2/2/88.

ture of almost universal susceptibility, especially where oral hygiene was poor (9-17). More recent basic, clinical, and epidemiologic study, however, has greatly sharpened our understanding of these diseases. This research has led to current perceptions of the diseases that differ from earlier ones, and that can be summarized as follows:

- 1. Only a small proportion of persons exhibit severe, widespread periodontitis. Mild gingivitis is common, and most adults demonstrate some loss of bony support and loss of probing attachment.
- 2. Gingivitis and periodontitis are associated with different bacterial flora. Gingivitis precedes periodontitis; however, not all sites with gingivitis later develop periodontitis.
- 3. Although usually related to age in population, periodontitis is not a natural consequence of aging.
- 4. Periodontitis is not the major cause of tooth loss in adults.

Periodontal disease is a generic term for a set of bacterial, inflammatory conditions of the supporting structures of the teeth. Until more specific interventions are developed, prevention is necessarily based on public education for oral hygiene and on professional cleaning at periodic intervals. This paper examines the scientific basis for these practices in light of recent research, and explores the related issue of targeting in the public health approach to control of gingivitis and adult periodontitis. Less prevalent periodontal conditions that do not appear preventable by public health means, such as localized juvenile periodontitis, are not included in this review.

Review of Recent Research_

"Severe" periodontitis usually means loss of attachment of 6 mm or more, and "generalized" periodontitis means an unspecified but considerable number of teeth affected. Epidemiologic evidence now suggests that generalized, severe periodontitis is unusual. This appears true even when oral hygiene is poor, gingivitis severe, and professional treatment limited (18-23). In populations with better oral hygiene, severe periodontitis is even less prevalent, though some degree of gingivitis is common (24-29). Initial reports from the 1985-86 National Oral Health Survey of Adults state that only 8 percent of American employed adults under age

Vol. 48, No. 4, Fall 1988

65 suffer from severe periodontitis, defined as at least one site where loss of attachment was 6 mm or more (30). The modern use of precise periodontal measurements, rather than the earlier indexes that did not employ probing, has led to these changes in perceptions of susceptibility.

The association between age and periodontitis still requires further study. Several reports have concluded that severe periodontitis is no more prevalent in older than in younger persons (26,28), and analyses of national survey data from 1971-74 found tooth retention to be as closely related to oral hygiene as it was to age (31). Severe loss of attachment, however, was found in 34 percent of persons over 65 in the National Survey of Adults, compared to only 8 percent of those under 65 (30). These differences between older and younger groups might be real, or they might reflect sampling differences and cohort effects. The direct relationship between age and periodontal diseases may reflect principally the results of long-term plaque accumulations (32,33), perhaps exacerbated by poorer plaque tolerance in aging tissues (34,35). Older people still appear to be a priority group for public health periodontal programs.

"The bacterial flora associated with gingivitis and periodontitis are not the same, though whether the conditions should be considered different diseases or not is of little practical importance for current prevention strategies."

The bacterial flora associated with gingivitis and periodontitis are not the same (36-41), though whether the conditions should be considered different diseases or not is of little practical importance for current prevention strategies. Bacteria associated with deep periodontal pockets are gram negative and anaerobic (42-45). While this finding is used to evaluate therapy and identify patients at risk (46,47), as yet there is no direct evidence to support an etiological role for these bacteria in periodontitis. There are even disagreements among authorities over whether the gram negative anaerobic bacteria in periodontitis represent an overgrowth of endogenous bacteria (48) or are exogenous infections (49). Gingivitis seems to be a nonspecific inflammatory process; over 70 different species have been associated with gingivitis (50). Defining the bacterial etiology of both diseases is a complex task, and is further complicated by the variety of individual reactions to infection (49,51). Moore et al. (52) suggested that destructive disease results from a colonization sequence, rather than just an increase in plaque mass or bacterial counts. Even though gingivitis does not always progress to periodontitis, meaning that under the Moore et al. hypothesis the colonization sequence does not develop, gingivitis still seems to be a necessary precursor of periodontitis. The conclusion is that periodontal disease prevention and management must still be based on nonspecific control of bacterial plaque deposits.

Periodontal disease has long been seen as the major cause of tooth loss over age 35, though evidence for this stock belief seems based largely on one report that reflected treatment practices in the early 1950s (53). But even at that time there was evidence that the most severe disease was found in relatively few persons (54). More recent data from several countries have shown that periodontal diseases account for few extractions at all ages, and cannot be considered as the major reason for tooth loss (55-58). By the time the last edentulous person from the "focal infection" era (59) has passed on, it can be anticipated that tooth loss from periodontal diseases will be limited to those relatively few teeth beyond redemption, and that mass extractions as routine dental treatment will have joined leeching in the archives of health care.

Public Education for Oral Hygiene _

Douglass and his colleagues used national survey data to demonstrate that oral hygiene levels are improving over time (60). They also showed that while the proportion of individuals with pockets (3 mm or greater) did not change between the national surveys in 1960-62 and 1971-74, there was a considerable shift among the remainder away from having gingivitis toward having no inflammation at all. From the evidence linking plaque deposits with gingivitis, it is a plausible hypothesis that improved oral hygiene is causing the reduction in gingivitis.

The traditional approach toward oral hygiene is that all plaque should be removed, an approach that must be questioned for several reasons. One pragmatic reason is that while few people are capable of removing all plaque, oral hygiene levels are improving anyway. A corollary is whether a plaque-free condition, even if possible, is really necessary. If plaque forms naturally, it seems that some level of plaque must be compatible with oral health. To explore this issue, data from the NHANES I national survey were analyzed to compare people of all ages who had 25 or more teeth present (31). Results showed that oral hygiene levels at all ages were remarkably similar, suggesting that an oral hygiene level that corresponds to OHI-S scores of 0.3-0.6 might be compatible with tooth retention throughout life

A further aspect of oral hygiene practices is that dental floss is not popular with many people (61-64). Should this be a concern in dental health education of the public? While some form of interdental cleaning is usually considered necessary to maximize the efficiency of tooth cleaning, the evidence that floss is the best way to do this is mixed (61,65-68). Interdental brushes have been reported as preferred and more efficient, especially for those with interdental spaces (61). Actually, there is no clear evidence that all persons need to practice interdental cleaning to maintain periodontal

health, though it is likely that some will.

Public educational programs to control periodontal diseases have been based upon the assumption that gingivitis progresses to periodontitis, and is therefore worth preventing. But gingivitis does not always progress to periodontitis, so it is difficult to see gingivitis by itself as a public health problem. The notion of periodontitis as a public health problem is probably more acceptable, being widely prevalent even if not always severe. However, prevention of periodontitis is still necessarily based upon regular plaque removal, so while the goal is to prevent periodontitis rather than gingivitis, the nonspecific plaque control required is still the same.

Professional Care

The beneficial effects of regular professional cleaning, from intervals of two weeks to four months, have been documented in children (69-75) and in adults (76-80). The importance of regular professional maintenance care in persons who have received periodontal treatment has also been extensively reported (81-89). While there are some differences on how meticulous oral hygiene needs to be during the maintenance phase of clinical periodontal treatment (87,88), no one disputes the importance of good oral hygiene in periodontal patients. Regular professional removal of plaque, and establishing conditions to retard its subgingival regrowth, remains the basis for clinical management of periodontal diseases (48,90).

Professional care to remove plaque deposits is a cumbersome and inefficient form of treatment, much like the medical treatment of infections around the turn of the century: keeping wounds clean and hoping that the patient would get better. While further research is likely in time to bring about more precise treatment, nonspecific plaque removal is the best we can do with current knowledge. And even with more rational treatment, keeping the wound clean (i.e., plaque removal) will remain a sound treatment principle.

There are inherent problems in interpreting the results of long-term studies of disease outcomes. Virtually all suffer from severe loss of patients for follow-up, thus introducing a threat of bias. There are also uncertainties about whether improvements in attachment levels (91,92) represent natural healing or simply tightening of gingival attachment, and the "random burst" hypothesis (93), which if valid could affect observed disease outcomes, has yet to be fully accepted (94-97). Despite the value of maintenance care among treated periodontal patients, there are reports of patients in periodontal practices among whom lack of professional care did not necessarily result in the progression of severe disease, either over a short period (98) or the long term (99,100). Problems with interpreting clinical studies are exemplified by the data in Table 1, taken from a study that concludes that frequent maintenance is necessary for favorable periodontal outcomes (101). These data indicate that many pockets did not progress or even apparently healed in the absence of professional treatment; certainly the data do not support a univer-

TABLE 1
Progression in Probing Depth in 44 Patients in the Absence
of Periodontal Treatment: Average Time of 5.25 Years
Between Examinations (101)

Pinet Prom	Probing Depth (mm) Second Exam	Chara
First Exam	Second Exam	Change
1–3	1-3	72.9% same
	4–6—	
		27.1% deteriorated
	7+	
4-6	1-3	
		>91.4% same/better
	4-6	
	7+	8.6% deteriorated
7+	1–3	
		≻68.4% improved
	4-6	•
	7+	——31.6% same

sal need for regular professional care. In addition, if constant professional cleaning really is necessary to control periodontal diseases at the community level, everyone must become a patient for life—clearly not a feasible approach, and one philosophically out-of-tune with today's encouragement of more personal responsibility for individual health status.

Goals for Community Periodontal Health.

The goals for programs of education and treatment, and how to achieve them, need to be defined carefully against the background of (a) uncertainty about the natural history of the periodontal diseases, (b) improving levels of oral hygiene, and (c) the inability to predict susceptibility to periodontitis (47,102-104). Goals perhaps could be expressed in practical terms of tooth retention and the maintenance of a functioning dentition, rather than in the more utopian terms of low gingivitis or low plaque. Achievement of such goals is likely still to require regular professional care for susceptible persons, although those less susceptible may not suffer if they receive less regular care. Such goals also imply acceptance of a certain level of gingivitis, and perhaps even minor pocketing, as compatible with an esthetic and functioning dentition.

The implications of recent periodontal research relate principally to two issues: (a) more limited susceptibility to severe disease than previously thought, especially among older people, and (b) the continuing scientific justification for maintaining good oral hygiene. If dental public health is to be efficient in its periodontal programs, however, targeting will need to be employed. While this philosophy is now well accepted, its practical application is frustrated by the absence of screening methods. In the meantime, care services could primarily be directed at the institutionalized and handicapped who have evidence of disease severe enough to threaten a functional dentition, with ambulatory low-income persons and handicapped less-sus-

Vol. 48, No. 4, Fall 1988 255

ceptible persons as a second priority level. Education to promote a satisfactory level of personal oral hygiene should be directed at groups most susceptible to periodontitis, of which the most consistently identified are lower socioeconomic groups of all races. The accompanying challenge, as public health workers well know, is that favorable oral hygiene behavior is not easily developed in these groups. But until reliable tests for determining individual susceptibility to severe periodontitis become available, priority for public health programs will have to be determined from epidemiologic data.

References

- 1. Dubos R. The mirage of health. New York: Doubleday, 1959.
- McKeown T. A sociological approach to the history of medicine. In: McLachlan G, McKeown T, eds. Medical history and medical care; a symposium of perspectives. London: Oxford University Press, 1971:1-16.
- 3. McNeil WH. Plagues and peoples. New York: Doubleday, 1976.
- **4.** Longmate N. Alive and well, medicine and public health from 1830 to the present day. London: Penguin, 1970.
- 5. Chilton NW. Some public health aspects of periodontal disease. J Am Dent Assoc 1950 Jan;50:28-33.
- Marshall Day CD. Epidemiology of periodontal disease. J Periodontol 1951 Jan;22:13-22.
- American Dental Association, Council on Dental Health. The role of dentistry in chronic illness. J Am Dent Assoc 1954 Jun;48:687-97.
- AAPHD Subcommittee on Preventive Periodontics. Periodontal disease in America: a personal and national tragedy. J Public Health Dent 1983 Spring;43:106-17.
- US Public Health Service, National Center for Health Statistics. Periodontal disease in adults, United States, 1960-1962, by Kelly JE, Van Kirk LE. USPHS pub no 1000, (Series 11, no 12). Washington, DC: Government Printing Office, 1965.
- UŠ Public Health Service, National Center for Health Statistics. Basic data on dental examination findings of persons 1-74 years, United States, 1971-1974, Kelly JE, Harvey CR. DHEW pub no (PHS) 79-1662, (Series 11, no 214). Washington, DC: Government Printing Office, 1979.
- 11. Waerhaug J. Epidemiology of periodontal disease—review of literature. In: Ramfjord SP, Kerr DA, Ash MM, eds. World workshop in periodontics. Ann Arbor: University of Michigan, 1966:181-211.
- Russell AL. Epidemiology of periodontal disease. Int Dent J 1967 Jun;17:282-96.
- Ramfjord SP, Emslie RD, Greene JC, Held AJ, Waerhaug J. Epidemiological studies of periodontal disease. Am J Public Health 1968 Jun;58:1713-22.
- Suomi JD. Prevention and control of periodontal disease. J Am Dent Assoc 1971 Dec;83:1271-87.
- 15. Chilton NW, Miller MF. Diagnostic methods and epidemiology of periodontal disease. In: Klavan B, et al. International conference on research in the biology of periodontal disease. Chicago: University of Illinois, 1977:94-118.
- World Health Organization. Epidemiology, etiology, and prevention of periodontal diseases. Geneva: WHO Tech Rep Series no 621, 1978.
- 17. Sheiham A. The epidemiology of dental caries and periodontal disease. Paper given at the Symposium on Prevention of Major Dental Disorders, Marabou, Sundbyberg, Sweden, June 16, 1979
- Cutress TW, Powell RN, Ball ME. Differing profiles of periodontal disease in two similar South Pacific island populations. Community Dent Oral Epidemiol 1982 Aug;10:193-203.
- Buckley LA, Crowley MJ. A longitudinal study of untreated periodontal disease. J Clin Periodontol 1984;11:523-30.
- Baelum V, Fejerskov O, Karring T. Oral hygiene, gingivitis, and periodontal breakdown in adult Tanzanians. J Periodont Res 1986;21:221-32.
- Löe H, Anerud A, Boysen H, Morrison E. Natural history of periodontal disease in man. Rapid, moderate, and no loss of attachment in Sri Lankan laborers 14 to 46 years of age. J Clin Periodontol 1986;13:431-40.

 Powell RN. The natural history of periodontal diseases. Ann R Aust Coll Dent Surg 1984 Oct;8:26-30.

- Ismail AI, Eklund SA, Burt BA, Calderone JJ. Prevalence of deep periodontal pockets in New Mexico adults age 27 to 74 years. J Public Health Dent 1986 Fall;46:199-206.
- 24. Halling A, Bjorn A-L. Periodontal status in relation to age of dentate middle-aged women. Swed Dent J 1986;10:233-42.
- 25. Hoover JN, Tynan JJ. Periodontal status of a group of Canadian adults. J Can Dent Assoc 1986 Sept;52:761-3.
- Hugoson A, Jordan T. Frequency distribution of individuals aged 20-70 years according to severity of periodontal disease. Community Dent Oral Epidemiol 1982;10:187-92.
- 27. Pilot T, Schaub RMH. Reappraisal of periodontal treatment needs [Abstract]. J Dent Res 1985;64(Spec Iss):260.
- Beck JD, Lainson PA, Field HM, Hawkins BF. Risk factors for various levels of periodontal disease and treatment needs of Iowa. Community Dent Oral Epidemiol 1984 Aug;12:17-22.
- Hughes JT, Rozier RG, Ramsey DL. Natural history of dental disease in North Carolina, 1976-77. Durham: Carolina Academic Press, 1982.
- More Americans are keeping their teeth. ADA News, Nov 3, 1986.
- Burt BA, Ismail AI, Eklund SA. Periodontal disease, tooth loss, and oral hygiene among older Americans. Community Dent Oral Epidemiol 1985 Apr;13:93-6.
- 32. Page RC. Periodontal diseases in the elderly: a critical evaluation of current information. Gerontology 1984;3:63-70.
- Mandel ID, Gaffar A. Calculus revisited. J Clin Periodontol 1986;13:249-57.
- Anerud KE, Robertson PB, Löe H, Anerud A, Boysen H, Patters RM. Periodontal disease in three young adult populations. J Periodont Res 1983;18:655-68.
- 35. Van der Velden U. Effect of age on the periodontium. J Clin Periodontol 1984;11:281-94.
- Page RC, Schroeder HE. Periodontitis in man and other animals. Basel: Karger, 1982.
- Moore LVH, Moore WEC, Cato EP, Smibert RM, Burmeister JA, Best AM, Ranney RR. Bacteriology of human gingivitis. J Dent Res 1987 May;66:989-95.
- McHugh WD, et al. Workshop background paper. In: Kakehashi S, Parakkal PF, eds. Proceedings of the state of the art workshop. J Periodontol 1982 Aug;53:475-501.
- 39. Polson AM, Goodson JM. Periodontal diagnosis; current status and future needs. J Periodontol 1985 Jan;56:25-34.
- 40. Newman MG. Current concepts of the pathogenesis of periodontal disease: microbiology emphasis. J Periodontol 1985 Dec;56: 734-9.
- 41. Page RC. Current understanding of the aetiology and progression of periodontal disease. Int Dent J 1986 Sept;36:153-61.
- Slots J. The predominant cultivable microflora of advanced periodontitis. Scand J Dent Res 1977; 85: 114-21.
- Tanner ACR, Haffer C, Bratthall GT, Visconti RA, Socransky SS. A study of the bacteria associated with advancing periodontitis in man. J Clin Periodontol 1979;6:278-307.
- 44. Bragd L, Dahlen G, Wilkstrom M, Slots J. The capability of Actinobacillus actinomycetemcomitans, Bacteroides gingivalis, and Bacteroides intermedius to indicate progressive periodontitis; a retrospective study. J Clin Periodontol 1987 Feb;14:95-9.
- Slots J. Bacterial specificity in adult periodontitis; a summary of recent work. J Clin Periodontol 1986 Nov;13:912-7.
- Greenwell H, Bissada NF. Variations in subgingival microflora from healthy and intervention sites using probing depth and bacteriologic identification criteria. J Periodontol 1984 Jul;55:391-7.
- Listgarten MA, et al. Failure of microbial assay to reliably predict disease recurrence in a treated periodontitis population receiving regularly scheduled prophylaxes. J Clin Periodontol 1986;13:768-73.
- 48. Theilade E. The nonspecific theory in microbial etiology of inflammatory periodontal diseases. J Clin Periodontol 1986 Nov;13:905-11.
- 49. Genco RJ. Highlights of the conference and perspectives for the future. J Periodont Res 1987;22:164-71.
- 50. Moore WEC, Holdeman LV, Smibert RM, Good IJ, Burmeister JA, Ranney RR. Bacteriology of experimental gingivitis in young adult humans. Infect Immun 1982 Nov;38:651-67.
- 51. Listgarten MA. A perspective on periodontal diagnosis. J Clin

- Periodontol 1986 Mar;13:175-81.
- 52. Moore WEC, Holdeman LV, Cato EP, Good IJ, Smith EP, Ranney RR, Palcanis KG. Variation in periodontal floras. Infect Immun 1984 Dec;46:720-6.
- 53. Pelton WJ, Pennell EH, Druzina A. Tooth morbidity experience of adults. J Am Dent Assoc 1954 Dec;49:439-45. Allen EF. Statistical study of primary causes of extraction. J Dent
- Res 1944;23:453-8.
- 55. Bailit HL, Braun R, Maryniuk GA, Camp P. Is periodontal disease the primary cause of tooth extraction in adults? J Am Dent Assoc 1987 Jan;114:40-5.
- Cahen PM, Frank RM, Turlot JC. A survey of the reasons for dental extractions in France. J Dent Res 1985 Aug;64:1087-93.
- 57. Ainamo J, Sarkki L, Kuhalampi ML, Palolampi L, Piirto O. The frequency of periodontal extractions in Finland. Community Dent Health 1984 Nov;1:165-72.
- Kay EJ, Blinkhorn AS. The reasons underlying the extractions of teeth in Scotland. Brit Dent J 1986 Apr;160:287-90.
- Weintraub JA, Burt BA. Oral health status in the United States: tooth loss and edentulism. J Dent Educ 1985 Jun;49:368-76.
- Douglass CW, Gillings D, Sollecito W, Gammon M. National trends in the prevalence and severity of the periodontal diseases. J Am Dent Assoc 1983 Sept;107:402-12.
- 61. Bergenholtz A, Olsson A. Efficacy of plaque removal using interdental brushes and waxed dental floss. Scand J Dent Res 1984;92:198-203.
- 62. Davis P. Reaction: consensus and contention. NZ Dent J 1984 Oct;80:125-7.
- 63. Silverstein S, Gold D, Heibran D, Nelms D, Wycoff S. Effect of supervised deplaquing on gingivitis and plaque [Abstract]. J Dent Res 1977;56(Spec Iss A):85.
- 64. Horowitz AM, Suomi JD, Peterson JK, Matthews BL, Vogelsong RH, Lyman BA. Effects of supervised daily dental plaque removal by children after 3 years. Community Dent Oral Epidemiol 1980 Aug;8:171-6.
- 65. Bergenholtz A, Brithon J. Plaque removal by dental floss or . toothpicks. An intraindividual study. J Ćlin Periodontol 1980;7:516-24.
- 66. Gisselson H, Bjorn AL, Birkhed D. Immediate and prolonged effect of individual preventive measures in caries and gingivitis susceptible children. Swed Dent J 1983;7:13-21
- 67. Granath LE, Martinsson T, Matsson L, Bilsson G, Schroder U, Soderholm B. Intraindividual effect of daily supervised flossing on caries in schoolchildren. Community Dent Oral Epidemiol 1979;7:147-50.
- 68. Carter HG, Barnes GP, Radentz WH, Levin MP, Bhaskar SN. Effects of using various types of dental floss on gingival sulcular bleeding. Virginia Dent J 1975;52:18-32.
- 69. Axelsson P, Lindhe J. The effect of a preventive programme on dental plaque, gingivitis and caries in schoolchildren. Results after one and two years. J Clin Periodontol 1974;1:126.
- 70. Axelsson P, Lindhe J. Effect of fluoride on gingivitis and dental caries in a preventive program based on plaque control. Community Dent Oral Epidemiol 1975 Aug;3:156-60.
- 71. Axelsson P, Lindhe J, Waseby J. The effect of various plaque control measures on gingivitis and caries in schoolchildren. Community Dent Oral Epidemiol 1976 Nov;4:232-9.
- 72. Kjarheim V, von der Fehr FR, Poulsen S. Two-year study on the effect of professional toothcleaning on schoolchildren in Oppergord, Norway. Community Dent Oral Epidemiol 1980 Dec;8:401-6.
- 73. Lindhe J, Axelsson P, Tollskog G. The effect of proper oral hygiene on gingivitis and dental caries in schoolchildren. Community Dent Oral Epidemiol 1975 Aug;3:150-5.
- 74. Hamp SE, Johansson LA, Karlsson R. Clinical effects of preventive regimens for young people in their early and middle teens in relation to previous experience with dental prevention. Acta Odont Scand 1984;42:99-108.
- 75. Hamp SE, Nilsson T, Faresjo T, Gamsater G. Relevance of social and behavioral factors in the evaluation of dental health care for schoolchildren. Acta Odont Scand 1984;42:109-18.
- 76. Lovdal A, Arno A, Schei O, Waerhaug J. Combined effect of subgingival scaling and controlled oral hygiene on the incidence of gingivitis. Acta Odont 1961;19:537-55
- Axelsson P, Lindhe J. Effect of controlled oral hygiene procedures on caries and periodontal disease in adults. J Clin Perio-

- dontol 1981;8:239-48.
- 78. Lightner LM, O'Leary TJ, Drake RB, Crump PP, Allen MF. Preventive periodontic treatment procedures: results over 46 months. J Periodontol 1971 Sept;42:555-61.
- 79. Suomi JD, Greene JC, Vermillion JC, Doyle J, Chang JJ, Leatherwood EC. The effect of controlled oral hygiene procedures on the progression of periodontal disease in adults: results after third and final year. J Periodontol 1971 Mar;42:152-60.
- 80. Suomi JD, Smith LW, Chang JJ, Barbano JP. Study of the effect of different prophylaxis frequencies on the periodontium of young adult males. J Periodontol 1973 Jul;44:406-10.
- 81. Becker W, Berg L, Becker BE. The long-term evaluation of periodontal treatment and maintenance in 95 patients. Int J Periodont Restor Dent 1984;4:54-71.
- 82. Hill RW, Ramfjord SP, Morrison EC, Appleberry EA, Caffesse RG, Kerry GI, Nissle RR. Four types of periodontal treatment compared over two years. J Periodontol 1981 Nov;52:655-62.
- 83. Knowles JW, Burgett FG, Nissle RR, Schick RA, Morrison EC, Ramfjord SP. Results of periodontal treatment related to pocket depth and attachment level-eight years. J Periodontol 1979 May:50:225-33.
- 84. Morrison EC, Ramfjord SP, Burgett FG, Nissle RR, Schick RA. The significance of gingivitis during the maintenance phase of periodontal treatment. J Periodontol 1982 Jan;53:31-4.
- 85. Nyman S, Rosling B, Lindhe J. Effect of professional tooth cleaning on healing after periodontal surgery. J Clin Periodontol 1975;2:80-6.
- 86. Pihlstrom BL, Ortiz-Campos C, McHugh RB. A randomized four-year study of periodontal therapy. J Periodontol 1981 May;52:227-42.
- 87. Ramfjord SP, Morrison EC, Burgett FG, Nissle RR, Schick RA, Zann GJ, Knowles JW. Oral hygiene and maintenance of periodontal support. J Periodontol 1982 Jan;53:26-30.
- 88. Rosling B, Nyman S, Lindhe J. The effect of systematic plaque control on bone regeneration in infrabony pockets. J Clin Periodontol 1976;3:38-53
- 89. Wilson TG Jr, Glover ME, Malik AK, Schoen JA, Dorsett D. Tooth loss in maintenance patients in a private periodontal practice. J Periodontol 1987 Apr;58:231-5.
- 90. Polson AM. The relative importance of plaque and occlusion in periodontal disease. J Clin Periodontol 1986;13:923-7.
- 91. Goodson JM, Tanner ACM, Haffajee AD, Sornberger CC, Socransky SS. Patterns of progression and regression of advanced destructive periodontal disease. J Clin Periodontol 1982;9:472-
- 92. Lindhe J, Haffajee AD, Socransky SS. Progression of periodontal disease in adult subjects in the absence of periodontal therapy. J Clin Periodontol 1983;10:433-42.
- 93. Socransky SS, Haffajee AD, Goodson JM, Lindhe J. New concepts of destructive periodontal disease. J Clin Periodontol 1984;11:21-32.
- 94. Imrey PB. Considerations in the statistical analysis of clinical trials in periodontitis. J Clin Periodontol 1986;13:517-28.
- 95. Ralls SA, Cohen ME. Problems in identifying "bursts" of attachment loss. J Periodontol 1986 Dec;57:746-52.
- 96. Morrison EC, Kowalski CJ. Discussion: clinical measurements of periodontitis. J Clin Periodontol 1986;13:456-8.
- 97. Blomqvist N. On the choice of computational unit in statistical analysis. J Clin Periodontol 1985;12:873-6.
- Harley A, Floyd P, Watts J. Monitoring untreated periodontal disease. J Clin Periodontol 1987;14:221-5.
- 99. Hirschfeld L, Wasserman B. A long-term survey of tooth loss in 600 treated periodontal patients. J Periodontol 1978 May;49:225-
- 100. McFall WT Jr. Tooth loss in 100 treated patients with periodontal disease. J Periodontol 1982 Sep;53:539-49.
- 101. Becker W, Becker BE, Berg LE. Periodontal treatment without maintenance; a retrospective study in 44 patients. J Periodontol 1984 Sept;55:505-9.
- 102. Haffajee AD, Socransky SS, Goodson JM. Clinical parameters as predictors of destructive periodontal disease activity. J Clin Periodontol 1983;10:257-65.
- 103. Sheiham A. Screening for periodontal disease. J Clin Periodontol 1978:5:237-45.
- 104. Löe H, Morrison EC. Periodontal health and disease in young people: screening for priority care. Int Dent J 1986 Sept;36:162-7.