The Prevalence of Periodontal Disease in Different Populations During the Circumpubertal Period

by

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CRUDE PREVALENCE DATA can be meaningful only if they can be related to the characteristics of the disease under study and to the characteristics of the population in which disease occurs. Previous speakers in this conference have provided insights which should be helpful in interpretation of the prevalence data to be presented here today. Dr. Lamb outlined the intricate processes of immune mechanisms in tissue, mechanisms which can protect or destroy those tissues; Dr. Mickelsen observed that the Recommended Dietary Allowances are arbitrary standards, not necessarily demarking the limit between ample and deficient (and reminded us of the dangers inherent in broad generalization from limited data); Dr. Socransky made it very plain that the oral flora is not uniform but widely variable in composition and pathogenicity from person to person and possibly from geographic place to geographic place; and Dr. Kleinberg demonstrated that oral plaque can vary from person to person, and even between one site and another within a given mouth. Each of these observations seems to be apropos directly to an understanding of known prevalences of periodontal disease in young and teen-age children here and in other parts of the world.

Most of the data to be presented here were gathered under the aegis of the National Institute of Dental Research, through use of the Periodontal Index.[1] This index is crude, in the sense that examiners are directed to record a condition as present only if it is clearly obvious. As a result the index places relatively little emphasis on minor conditions, and relatively great emphasis on disease in advanced states.[2] It has proved to be effective in distinguishing between population groups relatively free of, and groups afflicted with, deep destructive periodontal disease.[3] It can be used in situations where electricity is not available and where true sterilization of instruments is difficult or impracticable. Under these conditions, and in the course of the studies to be discussed here, the macrocharacteristics of disease as observed in the several populations of children are essentially the same as the characteristics of disease in adults. It is not necessary to go beyond the propositions that oral microbiota vary in occurrence and in pathogenicity, and that tissue response is quite as variable, to understand how an occasional child may show extensive tissue destruction with very little overt plaque or other sign of infection, or the converse—how an occasional child with heavy masses of plaque, calculus, or debris may exhibit essentially normal and healthy gingival and periodontal structures.

An opinion of John Oppie McCall has been cited by a previous speaker. From his writings it seems doubtful whether McCall considered periodontal disease in children to be different in any way from periodontal disease in adults. He seemed to infer that the two conditions were the same when he said,

> . . . It has been a rather common assumption that this disease [periodontoclasia] is characteristic of physical maturity and that we do not have to worry about it until the patient approaches middle age. I think we must now give thought however to the possibility that periodontoclasia, to a very real extent, has its origin in childhood. . . .[4]

CRUDE PREVALENCE

Mean PI scores for a series of populations of children, aged 5 to 19, are shown in Table 1. These data have been assembled from a variety of sources,[5–14] particularly the series of international nutrition surveys mounted by the Interdepartmental Committee on Nutrition for National Defense, but possess the common factor that all of the examinations were made by me or under the hand of examiners closely associated with me,* so that the differences exposed must be considered to be true differences, and not merely due to failure of independent examiners to agree.

The average scores shown in Table 1 may be interpreted as indications of the clinical status of the average child, on the basis of relationships between PI scores and clinical conditions originally established by comparing patient scores with diagnoses determined through detailed clinical examinations.[15] The clinical status of the average children in the populations described in Table 1 appear in Table 2. At all ages the typical child seen in the United States and in Burma, and in most of the children in Lebanon, exhibited essentially normal gingivae. The average refugee child aged 15 to 19 years in Lebanon exhibited a definite and extensive gingivitis. This was the finding for children 10 years of age or older in all of the other populations, except that the typical child in Vietnam or Burma aged 15 to 19 years had

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*Examiners were: United States, Russell; Malaya, G. W. Burnett; Lebanon, Russell; Ecuador, J. C. Greene and E. C. Leatherwood; Jordan, Leatherwood (with Ghazi Bekain); Thailand, Burnett and Leatherwood (with Pranee Sirikaya and Rajda Chandravejjsmarn); Colombia, C. J. Donnelly (with Domingo Medina Rivadeneira); Ethiopia, N. W. Littleton; Vietnam, Russell and Leatherwood (with Le Van Hien); Burma, Littleton (with Than Khin Maung). Each of the American examiners had been standardized directly with Russell.

TABLE 1
Periodontal Status of Children Aged 5-19 Years,
Various Surveys Employing the Periodontal Index

Population	Ages 5-9 N	Ages 5-9 Mean PI	Ages 10-14 N	Ages 10-14 Mean PI	Ages 15-19 N	Ages 15-19 Mean PI
			Male			
United States[5] (white)	3,368	.02	7,882	.10	2,196	.19
Malaya[6]	380	.08	368	.15	123	.16
Lebanon[7]—civilian	——		34	.12	39	.32
refugee	——		38	.27	11	.48
Jordan[9]—civilian	187	.17	136	.41	69	.57
refugee	209	.24	111	.66	48	.63
Thailand[10]	134	.22	139	.43	43	.59
Vietnam[13]—civilians	172	.25	126	.45	70	.93
Highlanders	22	.22	18	.37	25	.93
Burma[14]	44	.60	79	.58	48	1.21
			Female			
United States[5] (white)	3,314	.02	8,040	.08	1,835	.18
Malaya[6]	376	.10	260	.11	88	.11
Lebanon[7]—civilian	——		66	.25	31	.19
refugee	——		34	.31	14	.74
Jordan[9]—civilian	186	.20	163	.31	53	.37
refugee	201	.22	141	.36	62	.39
Thailand[10]	137	.24	160	.32	82	.31
Vietnam[13]—civilians	154	.17	166	.31	66	.51
Highlanders	13	.29	10	.30	19	1.10
Burma[14]	41	.51	80	.61	28	.55
			Male and Female Combined			
United States[5] (white)	6,682	.02	15,922	.09	4,031	.19
Malaya[6]	756	.09	628	.13	211	.14
Lebanon[7]—civilian	——		100	.21	70	.26
refugee	——		72	.29	25	.63
Ecuador*[8]	828	.12	715	.29	547	.38
Jordan[9]—civilian	373	.19	299	.36	122	.48
refugee	410	.23	252	.49	110	.49
Thailand[10]	271	.23	299	.37	125	.41
Colombia*[11]	——		——		115	.52
Ethiopia*[12]	61	.59	310	.67	198	.61
Vietnam[13]—civilians	326	.21	292	.37	136	.73
Highlanders	35	.25	28	.35	44	1.00
Burma[14]	85	.56	159	.60	76	.97

*No sex-specific data available in these publications.

TABLE 2
Interpretation, Periodontal Status of Children
Aged 5-19 Years, Various Surveys Employing
the Periodontal Index (Male and Female Combined)

Population	Clinical Status of the Average Individual Aged 5-9 Years	10-14 Years	15-19 Years
United States—white	normal	normal	normal
Malaya	normal	normal	normal
Lebanon—civilian	——	normal	normal
refugee	——	normal	gingivitis
Ecuador	normal	gingivitis	gingivitis
Jordan—civilian	normal	gingivitis	gingivitis
refugee	gingivitis	gingivitis	gingivitis
Thailand	normal	gingivitis	gingivitis
Colombia	——	——	gingivitis
Ethiopia	gingivitis	gingivitis	gingivitis
Vietnam—civilan	normal	gingivitis	incipient disease
Highlanders	gingivitis	gingivitis	incipient disease
Burma	gingivitis	gingivitis	incipient disease

reached the point where obvious destruction of deep supportive tissues was beginning to occur.

But this conference is probably not concerned so much with the average child as with the unusual child, the child in whom the onset of disease comes early in life and in whom tissue destruction is swift and severe, with an apparently exaggerated response to an apparently minimal presence of local irritating factors. Not all individual cases of severe gingivitis proceed to pocketing and loss of attachment. In our longitudinal study of children in the counties adjacent to Washington, D.C., teen-aged children who exhibited a frank periodontal pocket for the first time in a given examination had generally presented with marked gingivitis the year before; but during that previous year we had seen just as many children with gingivitis just as advanced whose condition the following year was either unchanged or markedly improved.[16]

Some of the publications from which the data of Tables 1 and 2 were drawn listed the proportions of children in whom clear and obvious periodontal pockets were observed. These proportions are shown in Table 3. Disease in this advanced state was seen in about 3 percent of white children aged 15 to 19 years seen in the United States, and in about 3.2 percent of a smaller sample of black children aged 10 to 19 years. At the other end of the scale, similarly advanced disease was seen in about one child in three aged 15 to 19 years in the Palestinian refugee camps in Lebanon. I was an examiner in each of these studies. Differences as great as these seem unaccountable unless one can postulate differences in the numbers and character of the attacking organisms.

Other investigators have seen patterns quite consistent with those just reported here. Stahl and Morris found gingival recession in 7 percent of Army engineers aged 17 to 19 years.[17] Bossert and Marks reported "active disease" in 2 percent of insurance company employees aged 16 to 19 years.[18] Mehta, Grainger, and Williams found "necrotic" disease or "periodontitis complex" in 7 percent of their group of Canadian civil servants aged 15 to 19 years.[19] Marshall-Day, Stephens, and Quigley discovered "chronic destructive periodontal disease" in 6 percent of males and 3 percent of females in Boston aged 13 to 15 years.[20] In more recent studies under the auspices of the World Health Organization, other observers have returned PI scores of the same magnitude as ours, or higher, for children of equivalent ages in India, Ceylon, Nigeria, Iran, and the Sudan; pocketing was seen by Ramfjord in boys as young as 15 years in India in a survey which used the PDI Index.[21]

Population Characteristics

Virtually every individual case in our series of studies was inflammatory in character, of the type usually called marginal gingivitis. Only one of the several World Health Organization examiners reported seeing individuals with "questionable evidence of periodontosis."[21]

In our United States children (Tables 1, 2, and 3), positive PI scores were more apt to occur in boys than in girls, aged 15 to 19 years, but when disease was present the condition was more apt to be severe in girls than in boys. Pocketing was observed in 6.8 percent of boys and in 9.8 percent of girls in this age group whose PI scores were something higher than zero.[5] Children were less apt to exhibit gingival or periodontal disease if they came from families or neighborhoods of relatively favorable socioeconomic status.[16, 22]

The strong and consistent relation between mouth cleanliness, however measured, and periodontal disease, however measured, needs little elaboration here. This relationship has been observed in every field study in which it has been taken into account.[23]

Table 3
Percentages of Children with Obvious Pocket Formation, Various Populations Surveyed by Means of the Periodontal Index (Males and Females Combined)

Population	*Percent with Pocketing at the Age of* 5-9 Years	10-14 Years	15-19 Years
United States—white	0.1%	1.0%	3.0%
black	0	3.2%*	
Burma	(under 15, about 2%)		6.0%
Thailand	0	0.3%	4.8%
Lebanon—civilian	——	5.4%	11.4%
refugee	——	9.7%	32.0%

*Data for ages 10-19 years.

(For United States children with signs of disease, females led males. At the above age groups, males included 0.8%, 2.8% and 6.8% of individuals with pockets; females 0.9%, 5.1% and 9.8%.)

Some other hypotheses which had been entertained at the outset could not be supported by field study findings. Apparent ethnic differences seemed to be explained by differences in mouth cleanliness.[24] Tooth by tooth studies of dental caries and periodontal disease in 2,000 United States adults led to the conclusion that the two diseases were independent of each other.[25] There was no marked relation between periodontal disease in children and use of a fluoride domestic water.[26] Most surprising was the failure to demonstrate any relation between nutritional deficiency and periodontal disease in a series of nutrition surveys focused upon ill-fed individuals and groups in such widely diverse geographical areas as Alaska, Ethiopia, Ecuador, Vietnam, Chile, Colombia, Thailand, or Lebanon.[27]

Nigeria—A Special Case

But any inference that his periodontal condition is independent of the general physical condition of the whole individual is difficult to accept in view of the findings of such investigators as Emslie[28] and Enwonwu[29] for Yoruba tribesmen in Nigeria. Malnutrition is common in children of the Yoruba villages; kwashiorkor is frequent, and there are high prevalences of such childhood afflictions as measles and diarrheal diseases. Typical necrotizing ulcerative gingivostomatitis was seen in as many as one child in four aged three to five years. This condition sometimes progressed to cancrum oris; more frequently the end result was destruction of the alveolar process prior to eruption of the permanent teeth. PI scores reported by Enwonwu* for children in the several regions of Nigeria are shown in Table 4. Periodontal tissues of the average Yoruba child aged 15 to 19 years in Western Nigeria[29] had deteriorated to an extent not

*Dr. Enwonwu's PI examination technics had been standardized directly with Russell.

TABLE 4
Summary: Periodontal Status of Individuals Aged 5-19 Years Examined in Nigeria, ICNND Survey, 1965[29]

Ages	*Specific Populations*				*All Groups*
	Western Nigeria	*Southern Nigeria*	*Eastern Nigeria*	*Military*	
5-9	0.97 (77)	0.97 (20)	0.86 (45)		0.94 (142)
10-14	0.96 (20)	1.04 (43)	1.06 (19)	0.95 (15)	1.01 (97)
15-19	1.82 (5)	1.51 (20)	1.43 (20)	1.18 (52)	1.33 (97)
5-19	1.01 (102)	1.14 (83)	1.04 (84)	1.13 (67)	1.07 (336)
	Number and Percent with One or More Pockets				
5-9	1 1.3%	0 .0%	0 .0%		1 0.7%
10-14	0 .0%	0 .0%	0 .0%	0 .0%	0 .0%
15-19	4 40.0%	3 15.0%	1 5.0%	5 9.6%	13 13.4%
5-19	5 4.9%	3 3.6%	1 1.2%	5 7.5%	14 4.2%

approached by white adults in the United States until the age of 45 years,[30] and "by age 20-29 years, most of the villagers are already approaching the terminal stages of periodontal destruction."[29] Such findings as these lead one to doubt the flat statement that such host factors as nutritional states can have no influence on the initiation or progression of periodontitis.

SUMMARY

Nevertheless, the most plausible hypothesis explaining these extremely precocious and severe instances of periodontitis in children in whom local factors are not prominent, the wide differences in prevalence between one child population and another, and the children in whom heavy and widespread plaque and calculus are associated with normal investing tissues, would seem to rest on the variable pathogenicity of plaque organisms, interacting with immune mechanisms which can defend or destroy the integrity of tissues. One of the limitations of epidemiological study of periodontal diseases, despite their obvious infectious nature, has been the necessity of dealing with them as though they were diseases of unknown etiology. It is to be hoped that the current emphasis on oral microbiology (as exemplified by Dr. Socransky's paper) will lead to technics and procedures which will permit the identification and study of the microbiota responsible for destruction of the periodontal tissues under the austere conditions which frequently obtain in the field.

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Discussion of Dr. Russell's Paper

Dr. Everett: I think this morning's procedure to a large extent will be devoted to a discussion of periodontosis. I think Dr. Russell used the term "classic periodontosis." Would you define the term?

Dr. Russell: That is that condition in which there is a great deal of destruction to periodontal tissue with an accompanying low set of conditions for local irritation. From the epidemiological standpoint and with the tools we now have, we may not be able to settle this question until some of you bright people can come up with a germ-free human. Because we always see plaque in every person after a very early age.

Dr. Everett: Now, the term periodontosis, you may remember, is a term that Orban and Weinmann introduced in a paper in 1942. The description of this entity is much older and was possibly originated in 1920 or 1921. At that time it was going under several names.

Gottlieb did not say it was a degenerative disease. This was something that Weinmann and Orban said. Therefore we have to define our terms. Gottlieb defined periodontosis as a type of disease that was different from marginal atrophy in that it occurred in the deeper areas of the periodontium. He never said that inflammation and function were not etiologic factors. Gottlieb only stated that this atrophy was a condition in which the cementum reacted differently to these irritants and that the organized periodontal fibers were replaced by a loose connective tissue. That is all he said.

Dr. Socransky: I would like to comment on Dr. Russell's suggestion that differences may exist in the microbiota in different parts of the world. I was extremely impressed by the photographs from Nigeria and it brings to my mind some samples we received from Nigeria from Dr. Emslie several years ago. We took these samples and injected them into some guinea pigs. As one might suspect, tremendous lesions were produced that were more rapidly progressive and destructive than you would see from samples obtained from people in this area. We isolated two different distinct organisms which are not ordinarily found in this part of the world.

I think some of the differences that can be shown between different populations may simply be differences in microbiota and not differences in tissue resistance.

Dr. Massler: We continue to seek simple answers to complex problems, either the microbiota or the hosts. Just one comment. You are pointing out that the small, almost invisible amount of plaque material might be as toxic or more so than the gross. We scratched some of this material into a rabbit's eye and this type of material blew the eye up, the big gunky portion did nothing. I think we are moving towards biological systems.

Dr. Mickelsen: Did you find any correlation between the calcium intake and existence of periodontal disease? You mentioned Vitamin C.

Dr. Russell: We had two types of data, biochemical determinations, vitamin, carotene, riboflavin, thiamin and that sort of thing. Then we had diet assays. We could make no correlation between any of these and caries or periodontal disease.

In caries this doesn't mean much because when we count DMF teeth we are looking at the accumulated signs of caries over a lifetime.

But PI is at least responsive to changes. You can reduce any PI score to zero in the hands of a man like Paul Baer.

So there should be some response to a change and we just didn't see it. As you know many of these biochemical determinations have a great big plus-minus error attached to them.

But nonetheless something should have come through consistently and it didn't. The only time Vitamin C was statistically associated with periodontal disease was in Ethiopia. The level was only $P < .05$ and this on the basis of about 1,200 people. With a sample like that, the probable border line was $< .05$, so you don't put much dependence on it.