Periodontal Index Development for Longitudinal Studies

The Development of A New Periodontal Index for Longitudinal Epidemiological Studies of Periodontal Disease

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ABSTRACT

To develop a new periodontal index for longitudinal studies of the periodontal disease was decided following a critical review of existing knowledge. As the first phase of index development, this study was to determine the inter- and intra-observer variation in assessing the parameters of gingival stippling, gingival redness, gingival bleeding, pocket pus, gingival ulceration, gingival recession level and pocket depth, as well as to determine the inter-parameter relationship. Each of three observers examined these parameters on buccal periodontal tissues of upper first or second molar in 126 dental emergency patients and re-examined them in 44 subjects who returned about a week later. The results indicate that the majority of study subjects did not have gingival stippling, but presented various degrees of gingival redness and epithelial detachment. In very few cases only was pus or bleeding observable. No gingival ulceration was observed. Inter- and intra-observer agreement was very good for gingival stippling and gingival redness estimates, but was rather poor for gingival bleeding estimate and pocket pus judgement when subjects with positive signs only were considered. For the measurements of gingival recession and pocket depth, observer variation was greatly reduced by increasing the number of severity categories. There was a weak, though statistically significant, relationship among the parameters. To conclude, gingival stippling, gingival redness, gingival recession and pocket depth are the four parameters suitable for further development of the periodontal index.

Résumé

L'examen critique de l'état actuel des connaissances sur la parodontopathie s'avérait nécessaire de développer un nouvel indice pour les études longitudinales sur la dite maladie. Dans la première phase du développement de l'indice, notre travail consistait tant à déterminer les sept paramètres choisis qu'à déterminer le rapport parmi les paramètres ci-mentionné. Chacun des trois observateurs avait examiné ces paramètres sur les tissus parodontaux buccaux de la première ou la seconde molaire supérieure chez les 126 patients d'urgence et avait réexaminé les mêmes paramètres chez les 33 sujets qui revenaient environ une semaine plus tard. Les résultats indiquent que la plupart des sujets n'avaient pas de pointillé gingival, mais montraient de différents degrés de la rougeur gingivale et du décollement épithélial. Dans très peu de cas, seuls le pus et l'hémorragie étaient observables. Aucune ulcération gingivale n'était observée. L'accord des observateurs était jugé très bon pour les estimations du pointillé gingival et de la rougeur gingivale, mais plutôt médiocre pour l'estimation de l'hémorragie gingivale et le jugement du pus de poche lorsque seuls les sujets avec signes positifs étaient considérés. En ce qui a trait à la classification de la régression gingivale et de la profondeur de poche, la variation des observateurs s'était grandement réduite au fur et à mesure que le nombre des catégories graves augmentait. Il y avait un rapport faible, quoique statistiquement significatif, parmi les paramètres. Notre étude nous aboutit à la conclusion suivante que le pointillé gingival, la rougeur gingivale, la régression

gingivale ainsi que la profondeur de poche sont les quatre paramètres appropriés pour le développement avancé de l'indice parodontal.

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Chapter 1

AIMS OF THE PROJECT

1.1 Specific aims of this study

The specific aims of this study are two folds:

a. To make an extensive and critical review of existing knowledge with respect to the epidemiological pattern, indices for measurement, various suspected etiological factors and possible preventive measures of the periodontal disease.

b. To conduct an initial phase of study which comprises three major aspects as follows:

   i. To select objective parameters which will represent, so far as possible, the whole range of periodontal health and disease.

   ii. To test, for each parameter estimate or measurement, the inter- and intra-observer variation, and the association in selected pairs of parameters.

   iii. With results from this study, to evaluate and comment on the validity of conclusions made by some investigators from their clinical trials.

1.2 Broad and final aims

a. Based on the results from the present study, parameters which allow a reasonable inter- and intra-observer agreement in estimation or measurement are selected for the development of a new periodontal index. Such an index should be specifically suitable for longitudinal epidemiological studies of periodontal disease. Therefore, it must satisfy so far as possible the following characteristics and requirements:

i. Simple to use with a minimum of instruments.

ii. The inter- and intra-observer variation for cohort studies must be both small and readily estimated.

iii. The index must include parameters which adequately describe both minor and major disease conditions.

iv. Each component periodontal parameter in the new index should be weighted through field studies of the natural development of periodontal disease, so that the final index has prognostic value.

b. To employ this periodontal index for longitudinal studies of periodontal disease so that more direct association between suspected etiological factors and periodontal tissue conditions can be studied. The index will also permit better evaluation on the effects of various preventive measures against the periodontal disease -- the final goal.

Chapter 2

CRITICAL REVIEW OF EXISTING KNOWLEDGE

2.1 Introduction

There are two main areas of investigation indicated in the epidemiology of periodontal disease. The first area describes the distribution of the periodontal disease in terms of age, sex, race, geography etc. The second area involves explanation of patterns of distribution of the disease in terms of causal factors.

Most current information concerning the distribution of periodontal disease in population groups has been collected following the introduction of various indices for measuring the prevalence and severity of the periodontal disease. Some epidemiological findings cast doubt on traditionally held significance of nutrition, systemic physical condition, malocclusion and trauma from occlusion in the initiation and progression of periodontal disease. These data also contradict the results from clinical trials relating various nutritional and dietary factors to periodontal disease.

A critical and extensive review of current knowledge is presented in Appendix I. It presents currently acceptable knowledge concerning the epidemiological pattern of the disease as well as some contradictory conclusions existing among clinical impression, clinical trials and population studies with respect to the etiological significance of nutrition and general physical condition in periodontal disease. When possible, such discrepancy is explained in terms of the study designs, data treatment, interpretation of the results and so on.

In this section, an attempt is made to summarize the overall implication (rather than conclusion), from the review. Effort is made to present information for which sufficient knowledge is available and to point out some areas for which only limited or no knowledge exists. For detailed review and the sources of information, the reader is advised to refer to Appendix I.

2.2 Morbidity and severity data for periodontal disease

The terminology of incidence, prevalence and severity has been greatly abused and misused in the literature. There are extremely little data about the incidence rate of periodontal disease. When incidence rate is mentioned in the articles, more often than not, it is actually prevalence rate.

If deviation from perfect periodontal health is used as a yardstick, practically all human beings have periodontal disease. It is often useless to compare the prevalence of periodontal disease in different populations as prevalence of some kind will be close to 100 per cent in most populations. To increase the comparability, severity indices have been introduced and become the major measurements of periodontal disease. Severity of periodontal disease is sometimes expressed in prevalence rates of various stages of periodontal condition, such as without periodontal disease, with gingivitis only or apparent periodontal pocket formation.

Since periodontal disease, in general, progresses very slowly, like other chronic disease, it is very difficult to make a direct assessment on suspected causes based on prevalence data alone. Factors which initiate and promote the disease may also differ. While the latter factors may be studied with some degree of reliability, with the prevalence data, only incidence data can allow for any direct evaluation on the former factors.

2.3 Relationship between prevalence and severity of periodontal disease and suspected etiologic factors

2.3.1 Age

The gingivitis occurs at very early age. Its prevalence rate and severity reaches a first peak at early teens and levels off or declines thereafter until the late teens. In adult populations,both prevalence and severity increase slowly with increasing age. Exception to this general trend is not uncommon, especially during the period of mixed dentition when fluctuation of gingival tissue condition of the permanent teeth is substantial and severity assessment for gingivitis is comparatively difficult.

The destructive periodontal disease (appearance of true periodontal pocket) may actually occur as early as 10 years old. With practically no exception, its prevalence and severity continues to rise with increasing age. The average severity may not increase at the same rate from one age group to another. Indeed, a sharp break can be observed between the age group of 15 to 19 years and 20 to 24 years in some population. The relationship between age and chronic destructive periodontal disease is so strong that such relationship holds irrespective which parameter being employed to describe the severity of the disease, be it an index score (such as the Russell's PI), simply average pocket depth per subject or tooth, degree of alveolar bone loss, gingival recession rate, etc. Naturely, these parameters are well correlated with each other.

Despite an apparent relationship between periodontal disease and age, it is not certain whether age factor has any etiological significance in periodontal disease. From the fact that in U.S. adults of 55 to 64 years of age, about one-sixth shows no detectable periodontal disease and only two-fifths have true periodontal pockets, it seems unlikely that aging process itself is responsible for the initiation and progression of periodontal disease. Instead, age may be only an indirect indicator of the duration of existing etiological factor or factors which may or may not be related to age, and of the duration of disease's natural course since its inception.

2.3.2 Sex

Practically all studies conducted on well developed populations and some studies made on less developed populations reveal that periodontal disease occurs more frequently and severely among male than female adults. For younger age group, there seems to have no definite sex difference in the prevalence and severity of periodontal disease. In all studies, difference in periodontal condition between two sexes can be accounted for by their corresponding difference in oral cleanliness and cleansing frequency.

However, in under-developed populations, periodontal disease is worse among adult women than men. Though, in most cases, the disease is still worse among boys than girls. Differences between two sexes in oral cleanliness or cleansing frequency can not account for all their difference in periodontal condition. Whether worse physical condition due to frequent child-bearing in those female adults, as suggested by Waerhaug,[8] results in worse periodontal condition in themselves is not at all certain.

Considering inconsistent relationship between sex and periodontal disease, it seems reasonable to believe that the intermediate factor or factors rather than sex itself are directly responsible for the initiation and progression of the periodontal disease. The intermediate factors may be oral cleansing efficiency, systemic resistance as well as other factors yet to be discovered.

2.3.3 Geographic distribution and ethnic factor

Epidemiologic studies of the prevalence and severity of periodontal disease have been made on populations in more than 50 geographic areas. To compare most prevalence data as assessed by different investigators is, however, severely handicapped by great observer variation in disease measurement, even if same index is employed. According to prevalence and severity data collected by a research team (Epidemiology Branch, N.I.D.R., U.S.A.) as well as by persons who have worked with the same team, the more developed populations and regions are associated with less prevalence and severity of periodontal disease.

When differing ethnic groups reside in completely different geographic region, it is almost impossible to decide whether it is ethnic factor or region which is mainly responsible for differing periodontal health. However, studies have been made on various ethnic groups living within a well defined geographic region. All these studies indicate distinct prevalence and severity of periodontal disease among ethnic groups.

Factors associated with differing socio-cultural patterns among populations of global geographic regions as well as among various ethnic groups living within a defined geographic area may be responsible for the distinct prevalence and severity of periodontal disease. But the underlying determinants of disease prevalence and severity are far from understood. Oral cleansing frequency and cleanliness sometimes can account for almost all variation of periodontal condition among ethnic groups or populations living in distinct geographic areas, but can account for only small part of the variation in many other occasions, such as among four ethnic groups in Ceylon. Occasionally, oral cleanliness is totally unable to account for the different periodontal health between ethnic groups. In Trinidad, East Indians exhibit a slightly more severe periodontal disease but better oral cleanliness than Negroes. Similar phenomenon also exists among three major ethnic groups in Singapore.

2.3.4 Socioeconomic factor

Socioeconomic status which is based on years of formal education, family income, occupation or a combination of social and economic variables, has been consistently demonstrated to have an inverse association with the prevalence and severity of periodontal disease. Years of formal education, in comparison to family income and occupational classes, is more closely associated with severity of periodontal disease.

In more developed countries, most of the disease variation among various socioeconomic classes can be accounted for by the degree of oral cleanliness, toothbrushing frequency, dental awareness and professional dental care received. In less developed countries, malnutrition and other unknown factor or factors which are associated with poor living standard seem to be the more responsible variables than previously mentioned factors for worse periodontal condition among poverty-stricken low socioeconomic persons when compared with the higher status groups.

2.3.5 Urban-rural comparisons

Rural as compared to urban population tends to have greater prevalence and severity of periodontal disease. The extent of the difference in most cases is, however, little or negligible. Study in Trivantrum even reveals greater prevalence and severity of periodontal disease in urban than rural population.

Corresponding to the urban-rural difference in periodontal health, rural in comparison to urban population also accumulate more oral deposits.

Despite apparent relation between urban-rural factor and socioeconomic status and availability of professional dental care, no study has ever taken into consideration of these social factors when periodontal health is compared between urban and rural populations. In Indiana, "median school years of rural persons 25 years or older in county" rather than "the percentage of county residents classified as rural" is associated with the PI scores of periodontally involved children. It is therefore conceivable that the urban-rural difference in periodontal condition, if present, may drop to negligible levels if socioeconomic status is held constant.

2.3.6 Occupational hazards

Injuries of the periodontal tissue as a result of the occupational exposure to various specific agents are rather common. Most of such knowledge is however not obtained from specific periodontal surveys on various occupational groups.

Lead, bismuth, mercury and their compound in dust form can cause distinct pigmentation, without toxic symptoms, on inflammed marginal gingiva. Since the degree of gingival pigmentation of these metals depends to a large extent on pre-existing inflammation, it should not be considered as an accurate index for estimating the degree of exposure. Acute intoxication from these metals may result in acute ulcerative gingivostomatitis or even destruction of the underlying bone. Exposure to chemicals such as phosphorus, arsenic, chromium and benzene may cause necrosis of alveolar bone with loosening and exfoliation of the teeth.

Despite rapid industrial development and growing awareness and knowledge of occupational hazards in general during last two decades, most knowledge of oral manifestations of occupational hazards came from studies made during the first half of this century. Indeed, more studies are needed to examine possible harmful effect of periodontal tissue through occupational exposure to various new chemicals, especially organic chemicals.

2.3.7 Nutrition, diet and systemic conditions

2.3.7.1 Nutritional factors and dietary habit

World-wide nutritional survey conducted by Epidemiology Branch, U.S. National Institute of Dental Research as well as some studies conducted by individual investigator fail to demonstrate that the deficiency of any specific or group of nutrients, as determined by biochemical analyses, is significantly associated with the severity of periodontal disease. These negative findings may result from rather homogenous nutritional state and wide age range within each study population. Results from other studies, in Ceylon and Western Nigeria for example, suggest that protein malnutrition may be responsible for greater severity of periodontal disease among poverty-stricken people as compared with better nourished groups dwelling within the same geographic area. Protein malnutrition is an important responsible factor for the disease only in the absence of good oral hygiene regimen. It is therefore reasonable to believe that protein malnutrition is associated with the progression rather than initiation of the periodontal disease.

There are many clinical trials aiming to determine any harmful effect of carbohydrate supplements or beneficial effect from protein and various vitamin supplements. Unfortunately, results from these studies have not greatly substantiated or clarified the nature of relationship between dietary and nutritional factors and the prevalence or severity of periodontal disease as found in population surveys. While results from many clinical trials indicate that severity of periodontal disease may be reduced by withdrawal of sweet drinks, and by supplementation of protein and various vitamins, their validity is quite questionable due to following reasons. First, no evidence of deficiency in specific nutrient among study subjects. Second, possible failure in blind assessment. Third, relatively short study periods. Fourth, no control over diets in placebo or control group. Fifth, failure to control disturbance variables in the study, and sixth, incorrect methods of data analyses.

The only finding which seems to be more or less consistency throughout the dietary and nutritional studies is the reduction of gingival inflammation following vitamin C supplementation in subjects who initially have apparent vitamin C deficiency. Results from other studies do not allow any definite conclusion.

Although both population surveys and clinical trials so far fail to reveal precisely the etiologic importance of dietary and nutritional factors in the severity of periodontal disease, they however have demonstrated very clearly the fact that these factors are unlikely the major variable and certainly not the only ones responsible for the severity of periodontal disease.

There are nine population surveys which study the relationship between fluoride concentration in drinding water and periodontal health. The results show that most of the differences in periodontal health between communities having high and low fluoride concentration in domestic water are not great if they do exist at all. Hence, there is no adequate evidence to support the hypothesis that the use of fluoridated water results in improved periodontal health. The findings are, however, wholly incompatible with any hypothesis that the periodontal tissue of children or of adults are harmed by use of a fluoride-bearing domestic water.

2.3.7.2 Organic systemic disease

Many studies have been conducted to determine the interrelationship between various systemic diseases or conditions and the prevalence and severity of periodontal disease. The overall impression from these study results can be presented separately for those which have adequate knowledge and those which are not certain.

It seems certain that periodontal disease tends to be more severe in persons with following disorders or disease: poorly controlled diabetes, mongolism, other mental defects or retardation, cerebral palsy, increase of systolic blood pressure, peripheral arterial disease, retinal vascular changes, sickle-cell anemia, cyclic neutropenia, agranulocytosis, thrombocytopenia, leukemia, malignant neoplasms, liver cirrhosis, obstructive pulmonary disease, albuminuria and presenile osteoporosis.

The associations between periodontal disease and the following disorders or diseases are less convincing: mild or wellconrolled diabetes, glucose tolerance within normal range, coronary or hypertensive heart disease, elevated diastolic blood pressure, arteriosclerosis, allergic diseases, gastrointestinal disease, genitourinary diseases or other respiratory diseases.

This summary is by no means exhaustive. Knowledge in this area is far from complete. This is mainly the result of a failure in many previous investigations to study systemic disease on the basis of a disease entity or of a specific pathologic process involved. Another reason for insufficient knowledge is the incomplete history record about chronic systemic disease and chronic periodontal disease, which make the interpretation of their relationship very difficult.

2.3.7.3 Mental illness, psychological factors and personality

The investigations on the relationship between mental, psychological and personality factors and the periodontal health have been conducted on three kinds of populations: 1, emotionally disturbed subjects; 2, persons with periodontal disease; 3, special population such as military personnel or students.

A general impression from these studies is that when the study population consists of mentally ill and normal subjects, some degree of association between periodontal condition and emotional stress can be found. If the study subjects are from periodontal patients or specific population group, such association is less likely demonstratable. Therefore, it implies that only very severe emotional disturbance may contribute to the severity of periodontal disease. The mechanism by which mental factors affect periodontal tissue remains to be answered.

2.3.7.4 Puberty, menstruation, pregnancy and menopause

Puberty is frequently accompanied by an exaggerated response of the gingiva to local irritation and by an increase in calculus formation. Chronic destructive periodontal disease progresses rapidly after puberty.

There is a lack of good, controlled studies of the gingival changes during each menstrual cycle. The studies so far seems to indicate a periodic fluctuation of salivary bacterial counts, a variation of gingival exudate and changes in the nature of gingivitis as a function of the menstruation cycle. There is, however, a lack of quantitative data about the severity of gingivitis which demonstrate any definitive fluctuation in gingival disease in relation to the menstruation cycle.

Many studies have shown that pregnancy is associated with greater severity of gingivitis which can not be explained by corresponding increases in oral debris or calculus deposition. Increased severity of gingivitis seems to have no lasting deteriorating effect after delivery. There is no consistent finding with respect to the fluctuation in severity of gingivitis throughout pregnancy. Monthly assessment on a cohort group is needed to clarify this controversy. Gingivitis can be abolished and gingival normality maintained during pregnancy provided that gingival areas on the teeth are kept clean -- suggesting that pregnant state is a modifying rather than an initiating factor in gingivitis. Pregnancy is not associated with apical migration of the epithelial attachment.

Research concerning the possible effect of menopause on periodontal disease is far from completed. Based on very limited data, there is an indication of greater severity of periodontal disease among postmenopausal women than one might expect. More studies with better designs are needed.

2.3.8 Oral hygiene factors

2.3.8.1 Studies on severity of periodontal disease in relation to frequency and technique of oral cleansing

If oral cleansing methods are relatively standardized, more frequent cleansing is associated with a decrease in oral soft debris, better gingival health and lower PI score. There is no sufficient evidence that oral cleansing frequency is associated with alveolar bone loss or epithelial detachment. Withdrawal of oral cleansing always results in more accumulation of plaque and calculus and a greater degree of gingivitis. Re-administration of oral cleansing without long delay can restore the tissue back to their original condition. Proper oral cleansing methods, increased frequency of cleansing and periodical oral prophylaxis by dental professionals are all essential for removing both soft and hard oral deposits and for keeping healthy gingivae. A mouth rinse with water alone is not sufficient for oral cleansing, but antibiotic containing mouth rinse may help to maintain gingival health. The advantage of the automatic over manual toothbrush in oral cleansing is not substantiated.

2.3.8.2 Studies on severity of periodontal disease in relation to oral deposition

The prevalence and especially severity of periodontal disease is associated with the amount of oral debris and calculus. Such correlation is stronger if study population vary greatly in age, are more privileged in material needs and belong to higher social status. Oral debris seems to be more associated with gingivitis at younger age, whereas calculus emerged as stronger or even the only associated oral deposit with more severe form of periodontal disease as population become older.

2.3.8.3 The fallacies of using the Oral Hygiene Index and other similar indices as a measure of oral cleansing effect

Following the introduction of the Oral Hygiene Index as well as other similar indices, the term "oral hygiene" has been loosely used to denote both oral cleanliness and oral cleansing. Evidence shows that these indices actually measure not only oral cleansing effect, but also dietary, nutritional and constitutional factors. In addition, these indices may even measure part of the periodontal disease itself. Therefore, taking amount of dental deposits as an index of oral cleansing effect in the epidemiological studies of periodontal disease will result in mis-interpretation of the pathogenesis of this disease and in less effectiveness from preventive measure subsequently.

2.3.8.4 The fallacies of evaluating the association of periodontal disease with variables other than the OHI score and age by standardization of the latter two variables

Thereis a great deal of data to suggest that an individual's OHI score is, to some extent, a function of nutritional, dietary and systemic factors as well as an indirect measure of age, that subgingival calculus may be the result rather than the cause of the periodontal disease, and that individual's nutritional status also depends on age. To evaluate the relationship between periodontal disease and variables other than OHI score and age by standardizing the latter two variables, such as is being done in most epidemiological studies of periodontal disease has likely resulted in erroneous conclusions regarding the true relationship between periodontal disease and the suspected etiological factors.

2.3.9 Effect of smoking, betel nut chewing and alcohol drinking

There is now a general consensus that smoking tended to increase the prevalence of ulceromembranous gingivitis, simple gingivitis, active periodontal disease as measured by PI or PDI as well as destructive phase of periodontal disease. Corresponding to this association is a similar relationship existing between cigarette consumption and amount of soft and hard oral deposits. If factors of age and oral deposits are held constant, relationship between cigarette consumption and periodontal disease is greatly reduced in most cases and disappears completely in others.

Betel chewers in comparison to non-chewers tend to have higher prevalence and severity of periodontal disease and greater amounts of oral deposits. Betel consumption is a more destructive factor than is tobacco smoking as far as periodontal tissue is concerned. It may be one of the important factors responsible for higher prevalence and severity of periodontal disease in the Asiatic countries.

The effect of alcohol drinking on periodontal tissue has not been studied adequately. Based on very limited data, there seems to be little, if any, relationship between alcohol consumption and periodontal health.

2.3.10 Association with dental caries

The research data so far fail to demonstrate any simple clear-cut relationship between dental caries and periodontal disease. Neither the commonly held opinion that these two dental diseases are essentially the disease of dental plaque is supported. Quite contrary, research data suggest that these two dental disease have distinct epidemiologic patterns and do not share many common disease related variables, and that their association may vary from one disease stage to another, from age to age, and depend on particular human environments. In fact, the definite beneficial effect of fluoride intake to reduction of dental caries and lack of such effect on periodontal disease also implies that the prevalence of these two dental diseases are rather independent one from another.

2.3.11 Malocclusion, trauma from occlusion and oral habits

The degree of malocclusion seems to correlate with severity of periodontal disease, though the strength of the association varies from one feature of malocclusion to another. One must bear in mind that most malocclusion indices are designed for the purpose of orthodontic treatment and, therefore the weights assigned to various component features within each index may not be much meaningful in terms of their relative importance in the pathogenesis of periodontal disease.

Research on the possible effect of trauma from occlusion on periodontal tissue is greatly handicapped by the poor agreement as to the criteria of trauma from occlusion itself. Taking the over-all findings from various approaches, it seems unlikely that the trauma from occlusion can initiate periodontal disease. Whether it can facilitate the progression of the inflammation into periodontal ligament and causing pocket formation is not certain.

Oral habits such as tongue thrusting, bruxism, clenching, clicking have been suspected clinically for possible damaging effect on periodontal tissue. Yet practically all studies which compare the periodontal health of those with and those without these oral habits fail to find any such association. The only oral habit which is more consistently found to be associated with the periodontal disease is mouth breathing. It apparently causes some degree of gingivitis in the anterior teeth. Its mechanism is however not certain.

2.3.12 Microorganisms

It is only during last decade that a great deal of works have been directed to examine the role of microorganisms in the pathogenesis of periodontal disease in human. These studies have provided strong evidence that oral bacteriae are quantitatively associated with gingival inflammation. However, studies so far fail to demonstrate any single species or a group of microorganisms which are responsible for gingival inflammation. The mechanisms by which bacteriae cause destruction of the periodontal tissues is not yet understood. Whether or not the periodontal disease is transmissible through bateriae from one person to another remained to be answered. There is no single study so far to investigate the possible role of virus in the prevalence or severity of periodontal disease.

2.3.13 Iatrogenic (faulty dentistry) factors

Although numerous kinds of faulty dental restorations and orthodontic appliances have been condemned for causing various stages of periodontal disease, very few research has been done to clarify the exact nature. However, overhanging restorations, unsatisfactorilly constructed partial dentures and removable splints seem likely to have detrimental effect on periodontal tissues either through bacterial accumulation, or faulty force on periodontium or both.

2.4 Existing indices used for studies of periodontal disease

Since periodontal disease occurs in almost every subjects, therefore, several indices have been developed which take stages of the periodontal disease into consideration. No one should expect to find an index which will serve all purposes. Russell's PI is possibly adequate when population prevalences of periodontal disease are compared, provided that all the measurement is made by same person or persons who have achieved very reasonable observer agreement in measurement through intensive calibration.

Gingival Index from Loe and Silness seems to be suitable for clinical trials relating to alteration in gingivitis severity. Periodontal Disease Index by Ramfjord, Gingival-Periodontal Index by O'Leary et al and Gingival-Bone count by Dunning and Leach can improve the sensitivity of Russell's PI.

It is very questionable whether the arbitrary score assigned to each gingivitis severity, epithelial attachment level or alveolar bone loss is prognostically meaningful. Assigning three interval scores for epithelial attachment level or alveolar bone loss, instead of taking exact measurement such as millimeter, may also greatly reduce the measurement sensitivity. Giving higher scores for either of these two parameters and lower score for gingivitis and combining them into a single index is justified only if the progression of gingivitis indeed results in epithelial detachment and alveolar bone loss --- an assumption remaining to be proved. The observer reproducibility for some indices has been shown very poor and for others has never been evaluated.

Hence, it is apparent that none of the currently available indices is very suitable for longitudinal studies of the periodontal disease.

Corresponding to the development of periodontal indices and their application in the field studies, various indices for measuring the quality and quantity of oral deposits and irritants have been introduced. Like periodontal indices, the selection of an index depends on the type of study. In general, comparison between populations requires a simple index, such as Simplified Oral Hygiene Index. For clinical trials and other longitudinal studies, more sensitive and reproducible indices are required. As discussed in the section of oral hygiene factors, it is not very certain yet whether these indices have contributed to the understanding of the etiology of periodontal disease.

2.5 Conclusion from review of current knowledge

The descriptive data for periodontal disease which are collected with cross-sectional studies have contributed to the understanding of the epidemiological pattern of the disease. However, some of these data are contradictory to many traditionally held opinions and clinical findings with respect to the importance of dietary, nutritional and general physical conditions and diseases in the initiation and progression of periodontal disease. While undesirable study designs in most clinical trials and subjective clinical judgement are responsible for part of the contradictory findings, a major part of disagreement possibly is the result of inherent shortcoming of employing cross-sectional studies for investigating a chronic disease, such as periodontal disease. The etiological factors may not be actively present at time of study.

A longitudinal study should be able to provide more direct assessment on the cause-and-effect relationship. Furthermore, it will provide a natural history of the disease and exact relationship between periodontal disease and dental caries at various ages.

For these longitudinal studies, a periodontal index which is designed specifically for such purpose is essential.

Chapter 3

MATERIALS AND METHODS

The proposed periodontal index was to be developed in three phases. The objective of the first phase was to test the inter- and intra-observer estimate or measurement variations incurred with the use of a variety of parameters which describe the periodontal tissue alterations. Based on the results of phase one, only parameters which have a reasonable degree of inter- and intra-observer agreement were to be chosen for further study. The main purpose of the second phase was to assign weights to the selected parameters so that an index comprising the weighted parameter estimates would have severity implication. In principle, the parameter weights should be based on assessments such as histological findings which are more indicative of disease severity than are the usual clinical means of severity. Since such assessments are usually impossible to obtain, the alternative is to assume that the average of the clinical severity estimates made by a group of periodontologists would give the best available clinical estimates. To elaborate, a group of periodontologists estimate the periodontal disease severity in one tissue area of the subjects. The severity scores should range from clinically normal (0) to very severe (say, 10). At the same time, an independent investigator obtains measurements for the selected index parameters on the tissue area in question. The parameter weights can then be obtained by a principal component analysis or a multiple regression technique taking the selected parameters as the independent variables and average clinical severity estimates as the dependent variable.

The third phase was to validate the obtained weighted index. The point in question is whether periodontal tissue with high index scores have a poorer prognosis as judged by progressively impaired tooth function due to the tissue destruction. Hence, a longitudinal study is required to test that.

The present study covers the first phase only of index development, namely, the examination of inter- and intra-observer variation in measuring periodontal parameters. Since observer variation depends on the distribution of the examined parameter, it was originally planned to examine a group of people more or less representative of the general population with respect to periodontal disease. As such a population was not readily at hand, a practical alternative was to examine those who presented for dental emergency service in the dental faculty. Subjects from this group were asked to volunteer for the study. For the inter-observer variation assessment, three observers examined the same tissue area on the same subject and independently recorded the parameter scores or measurements. For the intra-observer error assessment, the same tissue area was examined by the same observers one week later. The interval between the two assessments was considered adequate to reduce observer memory from influencing the second assessment and in addition, subjects generally preferred to return for dental examination on the same day of the week.

3.1 Data Collection

3.1.1 Study Population

Over an eight-week period, every subject who presented at the emergency dental service at the Montreal General Hospital was asked to volunteer for the study. To increase the study population, ten third-year dental students also volunteered to act as subjects. To enter the study, each subject had to have at least one upper first or second molar which was not the tooth of chief complaint for emergency service.

3.1.2 Observers

Ideally, observers in this type of study should be thoroughly familiar with periodontal tissue anatomy, but not have clinical experience in periodontal treatment, so that objective estimates and measurements of tissue alteration rather than subjective diagnosis of the disease are recorded. Three observers (I. Habbi*, H. Kamenesky*, and H.C. Kuo**) served for the duration of the study. For convenience of description, they will be referred to as observers A, B, and C respectively. Before commencing the clinical examination the methods of data collection were explained and discussed among the three observers. Standardization (calibration) among observers in estimating and measuring each parameter was carried out for the first few subjects. Thereafter, the author regularly supervised the two observers with regard to the criteria of measurement for each periodontal parameter. This was to ensure, as far as possible, that the same criteria were used by all observers though they remained

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* Third year dental students at time of the study

** Present author

entirely free to decide what they considered the correct score. The observers saw each subject in the same order.

3.1.3 Site of Periodontal Tissue Measured

Only the buccal periodontal tissues of one upper first or second permanent molar were examined. To increase homogeneity of the tissue measurements, the right first upper molar was the first choice. If it was missing, required immediate treatment, or if it demonstrated cervical caries, a class V dental restoration which included the cemento-enamel junction,an alternate tooth was selected in the following preference order: left upper first molar, right upper second molar or left upper second molar.

3.1.4 The Selection of Periodontal Parameters

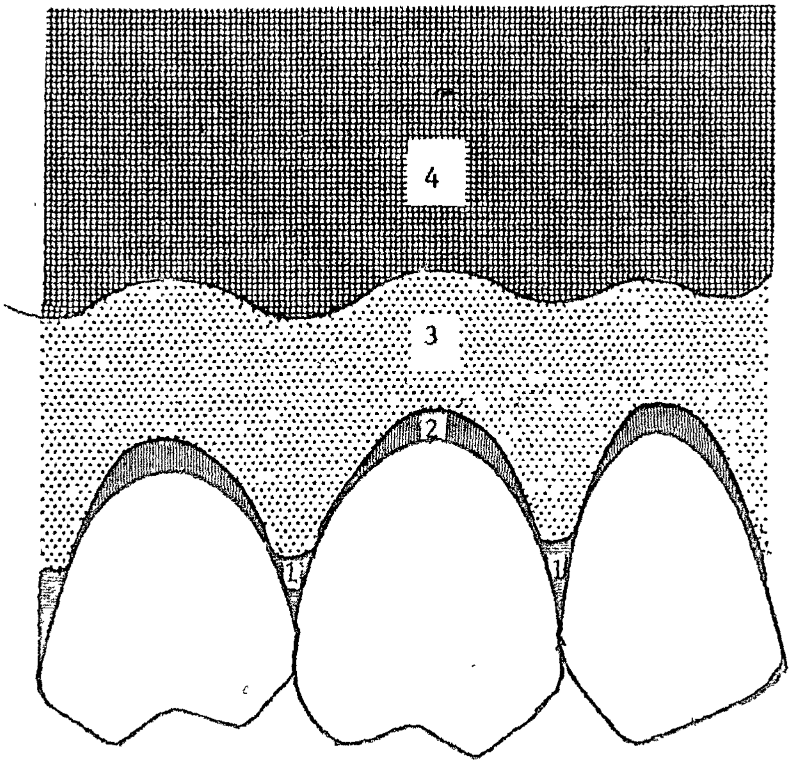
The definition of a basic gingival unit employed in the study was similar to that of Jackson (197) and is shown in Fig. 1.

Seven periodontal parameters were selected for measurement. These were:

a) Gingival stippling
b) Gingival redness and/or swelling
c) Gingival ulceration
d) Gingival bleeding
e) Pocket pus
f) Gingival recession level
g) Gingival or periodontal pocket depth

The algebraic sum of the last two measurements was taken as the epithelial attachment level. Table 1 shows the observer's recording sheet.

Fig.1 Demarcation of Gingival Units



1 ----- Papillary gingiva
2 ----- Marginal gingiva
3 ----- Attached gingiva
4 ----- Alveolar mucosa

Table 1

Observer Recording Form for Periodontal Conditions

Series number: __________ Examination 1 __________ 2 __________ Date ____________________
Name of subject ____ Observer ____________________
Age _______________ Sex _______________ Telephone ______________ Tooth examined ____________________

Parameter	Absent	Present		
		Buccal marginal gingiva		Attached gingiva
		Part	Whole	
Gingival stippling	- or 3			+ or 1.5 (part) ++ or 0 (whole)
Gingival redness and/or swelling	0	1	2	3
Gingival ulceration ✓	0	1	2	3
Gingival bleeding	0	1 (Mechanical pressure)	2 (Compressed air)	3 (Mouth rinse)
Pocket pus	- or 0		+ or 3	
Gingival recession level		__________ mm.		
Pocket depth		+ __________ mm.		
Epithelial attachment level		__________ mm.		

Gingival stippling, a sign of health, was scored (0-1.5) according to its presence on the whold or a portion of the attached gingiva. Complete absence of stippling, was scored as 3. For the parameters of gingival redness and/or swelling and gingival ulceration, absence was scored as 0, presence on a portion of the marginal gingiva as 1, on the whole marginal gingiva as 2, and if on the attached gingiva, 3 was the recorded score. The severity of gingival bleeding was determined according to its presence following a mouth rinse prior to the examination (3), following a stream of compressed air into gingival crevice or pocket (2), following external pressure on surface to be examined with the flat handle end of a Williams probe (1), (pressure equivalent to that which would cause paleness on a finger nail). In the case of no bleeding after mechanical pressure, the tissue was scored as 0. Since gingival bleeding was not reversible within a few minutes, only the first observer applied the various stimuli to the periodontal tissue, while each observer examined the result at the same time and recorded it without the knowledge of the others' scoring. Presence (3) or absence (0) of pus within gingival crevice or pocket was determined grossly following the application of mechanical pressure on the gingiva.

Gingival recession level and pocket depth were measured with a Williams periodontal probe at the mid-line of the buccal gingiva and recorded to the nearest one-half millimeter (mm). Calculus was removed, if necessary, to expose the cemento-enamel junction (C-E J). Three situations could arise with regard to the location of the gingival margin and epithelial attachment in relation to the C-E J. See Figure 2.

a. Both the gingival margin and the epithelial attachment were on enamel.

The cemento-enamel junction could not be felt by the periodontal probe. The depth of the gingival crevice was taken as "pocket depth" and its minus value as " gingival recession level". Epithelial attachment level was therefore 0.

b. The gingival margin was on the enamel, but the level of epithelial attachment was apical to the cemento-enamel junction.

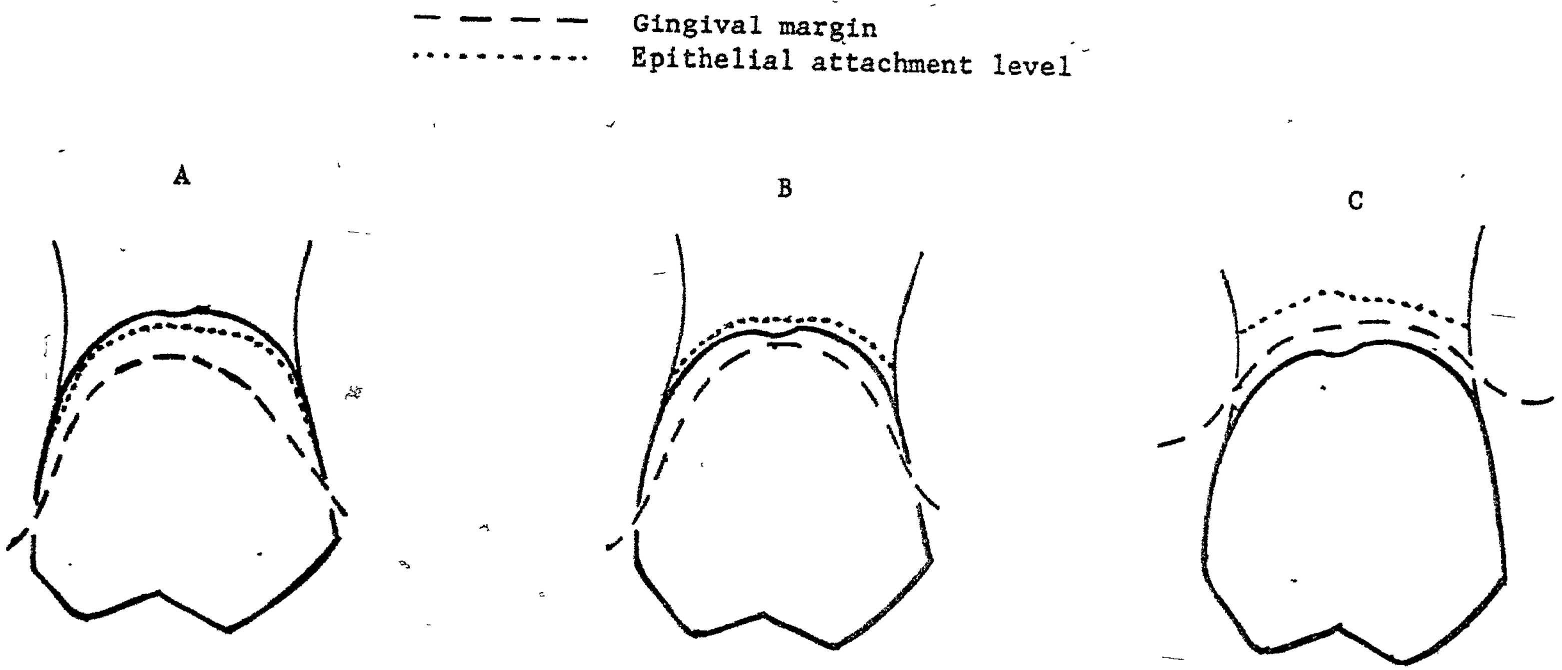
The distance from gingival margin to cemento-enamel junction was recorded in negative values as "gingival recession level", and the distance from the gingival margin to the epithelial attachment level or pocket bottom was the pocket depth.

c. The gingival margin was on the cementum.

The distance from cemento-enamel junction to gingival margin was taken as the "gingival recession level". The distance from the gingival margin to the epithelial attachment level or pocket bottom was recorded as the pocket depth.

Each patient was examined on a dental chair under artificial light. Mouth mirrors, Williams calibrated periodontal pocket probes, Ivory C.I. superficial scalers and compressed air were used routinely in the examinations. Following the first

Fig.2 Three Possible Locations of the Gingival Margin and the Epithelial Attachment Level in Relation to the Cemento-enamel Junction



A -- Both the gingival margin and the epithelial attachment level were on the enamel
B -- The gingival margin was on the enamel, but the level of epithelial attachment was apival to the cemento-enamel junction
C -- The gingival margin was on the cementum

examination, each subject was given an appointment for a second examination. The duration between two examinations was set as close as possible to one week. Therefore, each subject was to receive two assessments by all three observers.

Throughout the study period, about three-quarters of all invited subjects were only interested in receiving emergency treatment and did not want to be studied. Of those who volunteered for the study approximately one-fifth were not acceptable due to absence of previously outlined conditions. Thus, only a total of 126 subjects volunteered for and completed the initial examination. Although there was no adequate information with regard to their representativeness for all emergency patients or even for general population, based on their clinical picture, any such slight deviation from norm did not seem likely to create serious effect when they are used for studying observer variation in periodontal disease parameter estimation. There were 73 males and 53 females.

Of 126 subjects who received the first examination, only 44 or 34.9% returned and completed the second examination. These broke down as 28 males and 16 females. The age distribution of all subjects who were examined, those who completed the study and those who did not complete the study was shown in Table 2. Of the 82 subjects who did not return for the second examination, 45 were males and 37 were females. Proportionally, more males completed the study (38.4%) than the females (30.2%). Putting both sexes together, older subjects were more willing to be present for the second examination than were the younger subjects.

Table 2

Age Distribution of All Subjects Who Were Examined, Those Who Completed, and Those Who Did Not Complete The Study

	Age group (years)						
	16-25	26-35	36-45	46-55	56-65	65+	Total
	All examined subjects						
Number	55	28	22	10	8	3	126
Per cent	43.7	22.7	17.5	7.9	6.3	2.4	100.0
	Subjects who completed the study						
Number	16	8	8	6	5	1	44
Per cent	36.4	18.2	18.2	13.6	11.4	2.3	100.0
	Subjects who did not complete the study						
Number	39	20	14	4	3	2	82
Per cent	47.6	24.4	17.1	4.9	3.7	2.4	100.0
	Per cent within each age group						
Completed	29.1	28.6	36.4	60.0	62.5	33.3	34.9
Not completed	70.9	71.4	63.6	40.0	37.5	66.7	65.1

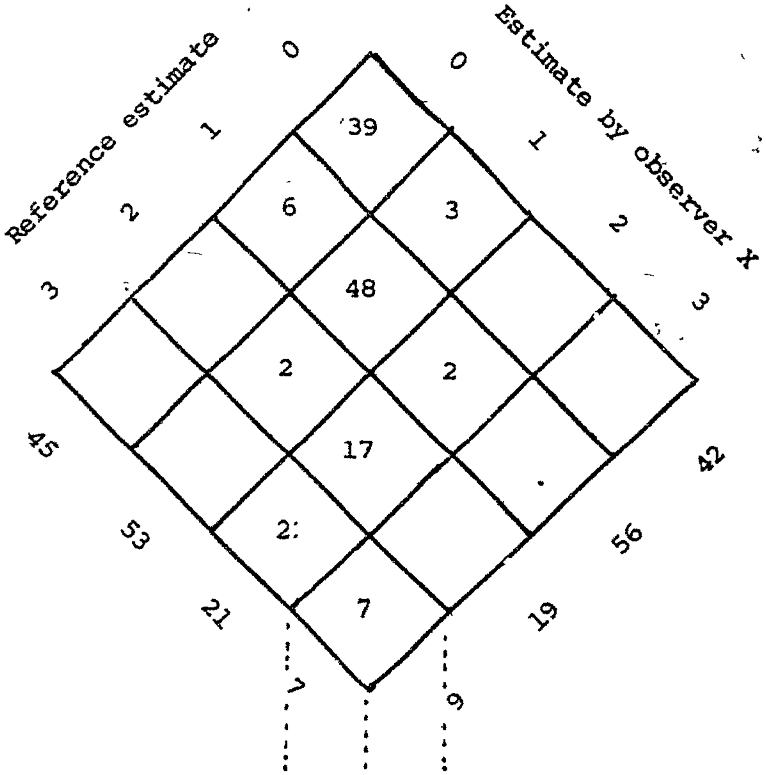
3.2 Method of data analyses

The frequency distributions of qualitative data, such as gingival stippling, gingival redness and/or swelling, gingival ulceration, tendency to bleeding and presence or absence of pus within gingival or periodontal pocket, were highly skewed and far from Gaussian (normal) distribution. Thus, regression analysis was not an appropriate method for the estimation of observer agreement in these parameters.

The inter-observer reproducibility or variation could be simply expressed as the percentage of times the same judgement or estimate was made by two independent observers. Since there were three observers in this study, the median of three observers' estimates on each parameter in the same subject was taken as the reference estimate for further comparisons. To describe the inter-observer variation, observer's scores were plotted against reference scores in a two-way distribution diagram as shown in Figure 3, such that the number and proportion of complete agreements and of various degrees of discrepancy could be estimated.

The assessment of inter-observer variation in the qualitative data was made by convertion to a quantitative analysis in terms of the average amount of information transmitted (AIT). The theory and technique of AIT analysis was developed for use in communication (90,387) but has been previously applied as an inverse measure of observer error (237) i.e., the greater the information transmitted, the lower the observer error(variation). AIT also has the merit of taking into account both sensitivity and consistency. Its calculation is based on the same two-way

Fig. 3 A Two-way Distribution Diagram for Each Parameter Showing Agreement or Disagreement of X Observer Estimate Against the Reference Estimate



Number of agreement or disagreement -------------------------	10	111	5
Percent of agreement or disagreement -----------------------	7.9	88.1	4.0
Deviation of X observer estimate from reference estimate ----	-1	0	+1

frequency distribution diagram (Figure 3). It is analogous to analysis of variance for quantitative data. Appendix II shows the details involved in an AIT analysis.

For quantitative data such as gingival recession, pocket depth and epithelial attachment level, the median of three observers' measurements was again taken as the reference measure. Both descriptive methods and AIT were employed to estimate the observer variation. Observer variation was also estimated in terms of the standard deviation of the distribution of variation and was calculated by the equation:

$$Se = (\Sigma d^2/2N)^{1/2}$$

where d was the difference between the replicate measurements, (in this case, the observer's estimate and the reference estimate) and N was the number of replicate measurements performed (417).

The above methods for estimating inter-observer variation for specific parameters were also employed to assess the intra-observer variation between first and second assessments. However, instead of plotting each observer's estimates or measurements against the reference ones, results from second assessment were plotted against the first ones for the same observer.

Again, an AIT analysis was employed to test the strength of association between selected pairs of parameters.

Chapter 4

RESULTS

4.1 Prevalence of each periodontal disease parameter

4.1.1 Prevalence of gingival stippling

The prevalence of gingival stippling in 126 subjects who received the initial examination is shown in Table 3. Based on the median of three observers' findings, gingival stippling appeared on the whole area of attached gingiva in only 5 (4%) of the subjects. The stippling was observed on a part of the attached gingiva in one-third of the subjects. Nearly two thirds (62.7%) of all subjects presented no gingival stippling. As shown in Table 3, gingival stippling judgement varied greatly from one observer to another. Whereas examiners B and C observed stippling in 53 and 55 subjects respectively, the presence of this parameter was reported in only 41 subjects by observer A. Gingival stippling, when present, existed mainly on the subpapillary area and only in some cases was it observable throughout the attached gingiva.

Comparing prevalence data of the 44 completed subjects with all those initially examined (Table 3), it is evident that the former group was fairly representative of the latter in that gingiva of nearly two-thirds of the subjects did not have observable stippling. Despite the fact that observers B and C independently detected in some cases, the appearance of stippling throughout the attached gingiva, in no case was such phenomenon agreed upon by both observers.

Table 3

Prevalence of Gingival Stippling as Estimated by Three Observers

Severity *	Observers							
	A		B		C		Median	
	number	%	number	%	number	%	number	%
	In all 126 subjects							
++	1	0.8	9	7.1	15	11.9	5	4.0
+	40	31.7	44	34.9	40	31.7	42	33.3
-	85	67.5	73	57.9	71	56.3	79	62.7
	In 44 completed subjects							
++	0	0.0	2	4.5	5	11.4	0	0.0
+	14	31.8	20	45.5	12	27.3	17	38.6
-	30	68.2	22	50.0	27	61.4	27	61.4

---

* ++: Present on whole area of the attached gingiva

+: Present on part of the attached gingiva

-: Completely absent

4.1.2 Prevalence of gingival redness and/or swelling

About one- third of all subjects who underwent the initial examination showed no gingival redness and/or swelling (Table 4). Based on median data of three observers' estimates, redness and/or swelling appeared on either part or whole marginal gingiva in 60% of all subjects. There were only 9 (7.1%) subjects in whom both the marginal and the attached gingiva showed redness and/or swelling.

In comparison to observer A and B, observer C tended to report higher severity for this parameter. It is noteworthy that in this study, not a single subject demontrated gingival swelling without concomitant redness of the tissue.

The initial prevalence of gingival redness and/or swelling in 44 subjects who completed the study is also shown in Table 4. Based on median data of the three observers' estimates, it appears that those who completed the study had a slightly lower prevalence of the parameter. As before, the three observers differed consistantly in their ability to estimate the extension of redness and/or swelling.

4.1.3 Prevalence of gingival bleeding

As mentioned in the section of Materials and Methods, prevalence and severity of gingival bleeding was examined after three degrees of gingival stimulation; namely, mouth rinse, compressed air into the gingival sulcus or pocket, or mechanical pressure on the gingival margin. It was unfortunate that in some cases, the parameter following each stimulation was not examined at the same time by all observers. Consequently, when bleeding did occur,

Table 4

Prevalence of Gingival Redness and/or Swelling as Estimated by Three Observers

	Observers							
Severity *	A		B		C		Median	
	number	%	number	%	number	%	number	%
	In all 126 subjects							
0	45	35.7	43	34.1	44	34.9	42	33.3
1	53	42.1	61	48.4	42	33.3	56	44.4
2	21	16.7	17	13.5	16	12.7	19	15.1
3	7	5.6	5	4.0	24	19.0	9	7.1
	In 44 completed subjects							
0	17	38.6	19	43.2	17	38.6	18	40.9
1	18	40.9	19	43.2	13	29.5	17	38.6
2	7	15.9	5	11.4	5	11.4	6	13.6
3	2	4.5	1	2.3	9	20.5	3	6.8

---

* 0: Absence of redness or swelling on the gingiva

1: Presence of redness and/or swelling on some part of marginal gingiva

2: Presence of redness and/or swelling on whole area of marginal gingiva

3: Redness and/or swelling extends to the attached gingiva the observer who did the last examination tended to report its appearance at a more severe stage. Table 5 brings out the result of this dilemma. In comparison to observer A and B, observer C recorded more severe gingival bleeding when it was present. Based on the same data, less than 10% of this study subjects had gingival bleeding which appeared mainly following mechanical pressure from a blunt hand instrument. Prevalence and severity data again showed that 44 subjects who completed the project were reasonably representative of those that began the study (Table 5).

4.1.4 Prevalence of gingival ulceration

During the study period, no subject demonstrated an ulcerated gingival surface detectable to the naked eye. However, ulceration may have been present on the lateral walls of gingival or periodontal pockets since this could not be verified by a visual examination.

4.1.5 Prevalence of pus within gingival or periodontal pockets

The presence or absence of pus was examined after the gingival margin had been subjected to a pressure from a blunt hand instrument. The median data of the three observers' estimates indicated that in only 5 (4.0%) of 126 subjects was pus observable (Table 6).

Table 6 also shows that the prevalence of gingival or periodontal pocket pus in the 44 subjects who completed the study was quite similar to that in all 126 subjects.

Table 5

Prevalence of Gingival Bleeding as Estimated by Three Observers

	Observers							
Severity *	A		B		C		Median	
	number	%	number	%	number	%	number	%
	In all 126 subjects							
0	114	90.5	114	90.5	115	91.3	116	92.1
1	9	7.1	10	7.9	6	4.8	7	5.6
2	2	1.6	1	0.8	3	2.4	2	1.6
3	1	0.8	1	0.8	2	1.6	1	0.8
	In 44 completed subjects							
0	41	93.2	38	86.4	42	95.5	40	90.9
1	2	4.5	4	9.1	0	0.0	2	4.5
2	1	2.3	1	2.3	1	2.3	1	2.3
3	0	0.0	1	2.3	1	2.3	1	2.3

* 0: Absence of gingival bleeding even under mechanical pressure

1: The gingiva starts to bleed following an application of mechanical pressure

2: The gingiva starts to bleed following an application of compressed air

3: Presence of gingival bleeding following mouth rinsing

Table 6

Prevalence of Gingival or Periodontal Pocket Pus as Judged by Three Observers

	Observers							
Pocket pus	A		B		C		Median	
	number	%	number	%	number	%	number	%
	In all 126 subjects							
Absent	120	95.2	117	92.9	124	98.4	121	96.0
Present	6	4.8	9	7.1	2	1.6	5	4.0
	In 44 completed subjects							
Absent	41	93.2	41	93.2	43	97.7	42	95.5
Present	3	6.8	3	6.8	1	2.3	2	4.5

4.1.6 Prevalence of gingival recession

Gingival recession, here defined as the distance from cemento-enamel junction to gingival margin, was expressed as positive when the gingival margin was apical to C-E junction and as negative when the margin was on coronal portion. Table 7 shows the prevalence of gingival recession in the 126 subjects who received the initial examination. The median data of three observers' estimates indicate that in 44 subjects (35%), the gingival margin was located on the crown, and in 33 cases (26.2%), the gingival margin was at the level of C-E junction. In the remaining 49 subjects (38.9%), the C-E junction was completely exposed. There was one subject whose gingiva had receded nearly 7 mm apical to the C-E junction. In an additional six persons the root was exposed for between 4.0 and 6.5 mm.

There seemed to be a considerable variation among the three examiners in identifying the C-E junction. Whereas examiner C reported 71 subjects whose gingiva was on the crown and 11 subjects whose gingival margin was on the C-E junction; these two situations were recorded by observer A in 31 and 46 cases and by observer B in 41 and 35 subjects respectively. The total number of subjects presenting with either of these two situations appeared very similar, being recorded as 77, 76, and 82 by observer A, B and C respectively.

Compared to the data from all initially examined subjects, the data from the group who completed the study indicated a greater amount of gingival recession (Table 7). Variation among ob-

Table 7

Prevalence of Gingival Recession as Measured by Three Observers

	All 126 subjects					44 completed subjects				
	By observers					By observers				
Gingival recess.	A	B	C	Median		A	B	C	Median	
(mm.)	no.	no.	no.	no.	%	no.	no.	no.	no.	%
-3.5	0	0	1	0	0.0	0	0	0	0	0.0
-2.0	1	1	5	2	1.6	0	1	2	1	2.3
-1.5	1	2	7	2	1.6	0	0	1	0	0.0
-1.0	13	14	37	16	12.7	3	4	8	5	11.4
-0.5	16	24	22	24	19.0	1	5	6	3	6.8
0	46	35	11	33	26.1	13	8	6	9	20.5
+0.5	14	11	12	13	10.3	6	4	1	4	9.1
+1.0	6	11	9	8	6.3	5	7	7	7	15.9
+1.5	6	7	5	8	6.3	4	3	3	4	9.1
+2.0	11	8	7	7	5.6	5	5	2	4	9.1
+2.5	1	0	1	1	0.8	0	0	1	0	0.0
+3.0	4	4	3	5	4.0	1	1	1	1	2.3
+3.5	0	0	1	0	0.0	0	0	1	0	0.0
+4.0	2	5	0	2	1.6	1	2	0	1	2.3
+4.5	1	0	1	1	0.8	1	0	1	1	2.3
+5.0	2	3	3	3	2.4	2	3	3	3	6.8
+6.0	0	0	1	0	0.0	0	0	1	0	0.0
+7.0	2	1	0	1	0.8	2	1	0	1	2.3

servers in identifying the C-E junction was quite apparent when assessment on the 44 subjects was considered alone. Whereas observer A, B and C respectively reported 17, 18 and 23 subjects with gingival margin located either on or coronal to the C-E junction, the total number of subjects whose tooth roots were exposed for 0.5 mm or less (including not exposed) was 23, 22 and 24 respectively for observer A, B and C. It appears that the variation among observers in locating the C-E junction was about ±0.5 mm.

4.1.7 Prevalence of gingival or periodontal pocket depth

The gingival or periodontal pocket was measured from the gingival margin to the level of the epithelial attachment or to the pocket bottom, disregarding the level of gingival margin on the tooth surface. The median data of three observers' measurements showed that in 53 of the 126 subjects (42%) who received the initial examination, sulcus or pocket depth was 1 mm or less (Table 8). Only in 11 subjects (8.7%) of those examined, were pockets deeper than 2 mm of which one subject had pockets of 4 mm in depth.

In comparison to all persons examined, the 44 subjects who completed the study had slightly deeper pockets (Table 8). Pockets deeper than 2 mm were diagnosed in 8 (18%) of these subjects.

As expected, there was some observer variation in the prevalence data. In general, observer C tended to obtain higher readings in measuring pockets of 2 mm or more than did observers A and B.

Table 8

Prevalence of Gingival or Periodontal Pocket as Measured by Three Observers

Pocket depth	All 126 subjects					44 completed subjects				
	By observers					By observers				
	A	B	C	Median		A	B	C	Median	
(mm.)	no.	no.	no.	no.	%	no.	no.	no.	no.	%
0.0	2	2	1	2	1.6	1	1	1	1	2.3
0.5	15	18	9	15	11.9	5	6	1	5	11.4
1.0	41	33	49	36	28.5	15	11	18	11	25.0
1.5	34	34	27	37	29.4	11	10	10	14	31.8
2.0	21	26	21	25	19.8	4	7	6	5	11.4
2.5	6	6	9	6	4.8	4	5	3	5	11.4
3.0	6	6	8	4	3.2	3	3	4	2	4.5
3.5	0	0	1	0	0.0	0	0	0	0	0.0
4.0	0	0	1	0	0.0	0	0	1	0	0.0
4.5	1	0	0	1	0.8	1	0	0	1	2.3
5.0	0	1	0	0	0.0	0	1	0	0	0.0

4.1.8 Prevalence of the epithelial attachment level

The epithelial attachment level measures the distance from the C-E junction to either the epithelial attachment or the periodontal pocket bottom. The prevalence data are presented in Table 9 for 126 subjects who received the initial examination and for the 44 subjects who completed the study. Summarized prevalence data are also shown in Table 10.

When the C-E junction is covered by the epithelium, it should be impossible to measure the epithelial attachment level, and in principle, a zero value should be recorded. In a few cases, however, the attached level was clearly located on the coronal portion of the tooth and therefore a value of -0.5 mm was assigned for detachment. Median data of the three observers' measurements (see summary table 10) showed that while only one-eighth of the subjects had no measurable epithelial detachment, less than one-eighth of all subjects had an epithelial detachment of greater than 3.0 mm. All 5 of the 126 persons with greatest detachment (greater than 6.0 mm) had completed the study.

Also shown in Table 10 is the great variation among observers in deciding whether epithelial attachment level was on or apical to the C-E junction. In contrast to observer A and B, observer C consistently recorded more subjects without a detectable epithelial detachment. Observer A was inclined to find detached epithelium on cementum.

Table 9

Prevalence of Epithelial Attachment Level as Measured by Three Observers

	All 126 subjects					44 completed subjects				
	By observers					By observers				
Epithel. attach.	A	B	C	Median		A	B	C	Median	
(mm.)	no.	no.	no.	no.	%	no.	no.	no.	no.	%
-0.5	3	4	2	1	0.8	0	2	0	0	0.0
0	5	11	44	16	12.7	1	3	11	5	11.4
+0.5	27	16	11	24	19.0	8	5	3	6	13.6
+1.0	23	26	10	19	15.1	7	4	4	4	9.1
+1.5	15	13	11	15	11.9	2	3	2	3	6.8
+2.0	16	18	15	16	12.7	6	7	5	6	13.6
+2.5	6	9	8	4	3.2	2	4	5	2	4.5
+3.0	7	8	8	11	8.7	5	6	3	9	20.5
+3.5	8	5	3	4	3.2	4	0	2	0	0.0
+4.0	3	1	3	3	2.4	2	1	2	1	2.3
+4.5	3	4	1	3	2.4	1	2	0	2	4.5
+5.0	2	2	5	2	1.6	0	1	2	0	0.0
+5.5	1	3	0	2	1.6	0	1	0	1	2.3
+6.0	1	0	1	1	0.8	0	0	1	0	0.0
+6.5	2	1	0	1	0.8	2	1	0	1	2.3
+7.0	1	1	1	1	0.8	1	0	1	1	2.3
+7.5	0	1	0	0	0.0	0	1	0	0	0.0
+8.0	0	1	2	1	0.8	0	1	2	1	2.3
+9.0	0	0	1	0	0.0	0	0	1	0	0.0
+9.5	2	0	0	1	0.8	2	0	0	1	2.3
+10.0	1	2	0	1	0.8	1	2	0	1	2.3

Table 10

Epithelial Attachment Level as Measured by Three Observers: Summarized Data

Epithel. attach.	Observers							
	A		B		C		Median	
(mm.)	number	%	number	%	number	%	number	%
	In all 126 subjects							
0	8	6.4	15	12.0	46	36.5	17	13.5
0.5-3.0	94	74.6	90	71.3	63	49.8	89	70.6
3.5-6.0	18	14.3	15	12.0	13	10.4	15	12.0
6.5+	6	4.8	6	4.8	4	3.2	5	4.0
	In 44 completed subjects							
0	1	2.3	5	11.4	11	25.0	5	11.4
0.5-3.0	30	68.2	29	65.9	22	50.0	30	68.1
3.5-6.0	7	15.9	5	11.4	7	15.9	4	9.1
6.5+	6	13.6	5	11.4	4	9.1	5	11.4

4.1.9 Summary of prevalence data

To summarize, the buccal periodontal tissue of the upper first or second molars in this study population showed the following characteristics: most attached gingiva did not have a stippled appearance and when present, it was located on the subpapillary areas; one-third of the subjects showed no gingival redness or swelling and in nearly two-thirds of the cases, part or all of the marginal gingiva was red in colour; such a colour change usually did not extend to the attached gingiva; there was no observable gingival ulceration and in only 4% of the cases was pocket pus observable; in more than nine-tenths of the subjects, gingival bleeding could not be induced with a mechanical pressure; the gingival margin was located on the coronal portion of the teeth in more than one-third of the subjects and on the C-E junction in an additional one-quarter of the subjects; in less than one-tenth of the subjects were the gingival or periodontal pockets deeper than 2 mm; and when the epithelial detachment was taken as an estimate of definitive destruction of periodontal tissue, one-eighth of the subjects showed no sign of epithelial detachment while in an additional 70% of the subjects was the detachment no more than 3 mm apically to the C-E junction.

It would appear that the study group had some degree of gingival inflammation without grossly observable bleeding, ulceration or pocket pus. The majority of the subjects demonstrated incipient destruction of the periodontal ligament as manifested by apical migration of the epithelial attachment, but only in a few cases did such destruction start to affect the masticatory function.

4.2 Inter-observer variation of parameter estimate or measurement.

The inter-observer variation for all parameters could be presented in two ways: one, by displaying a frequency distribution of the deviation of each observer estimate or measurement from the reference estimate or measurement which was the median of three observer estimates; and second, employing the statistics of the average amount of information transmitted as an inverse measure of observer error or variation. The statistic of measurement error or variation was also used to estimate inter-observer variation for gingival recession, pocket depth and epithelial attachment level due to their parametric nature.

4.2.1 Inter-observer variation of gingival stippling estimate

Inter-observer variation for the gingival stippling estimate in 126 subjects is shown in Fig. 4. Complete agreement of each observer's estimate with the reference estimate was 87% for observer A and B and 75% for observer C. In general, observer A tended to underestimate the gingival stippling, whereas observer C reported greater prevalence and amount of stippling. Similar inter-observer variation was observed in initial and final gingival stippling assessment on the 44 subjects who completed the study.

The crude impression of inter-observer variation was substantiated by the average amount of information transmitted (AIT), Table 11. The AIT based on the data from 126 subjects was 0.48, 0.67 and 0.43 for observer A, B and C respectively,

Fig. 4 Inter-observer variation of gingival stippling estimate in 126 subjects

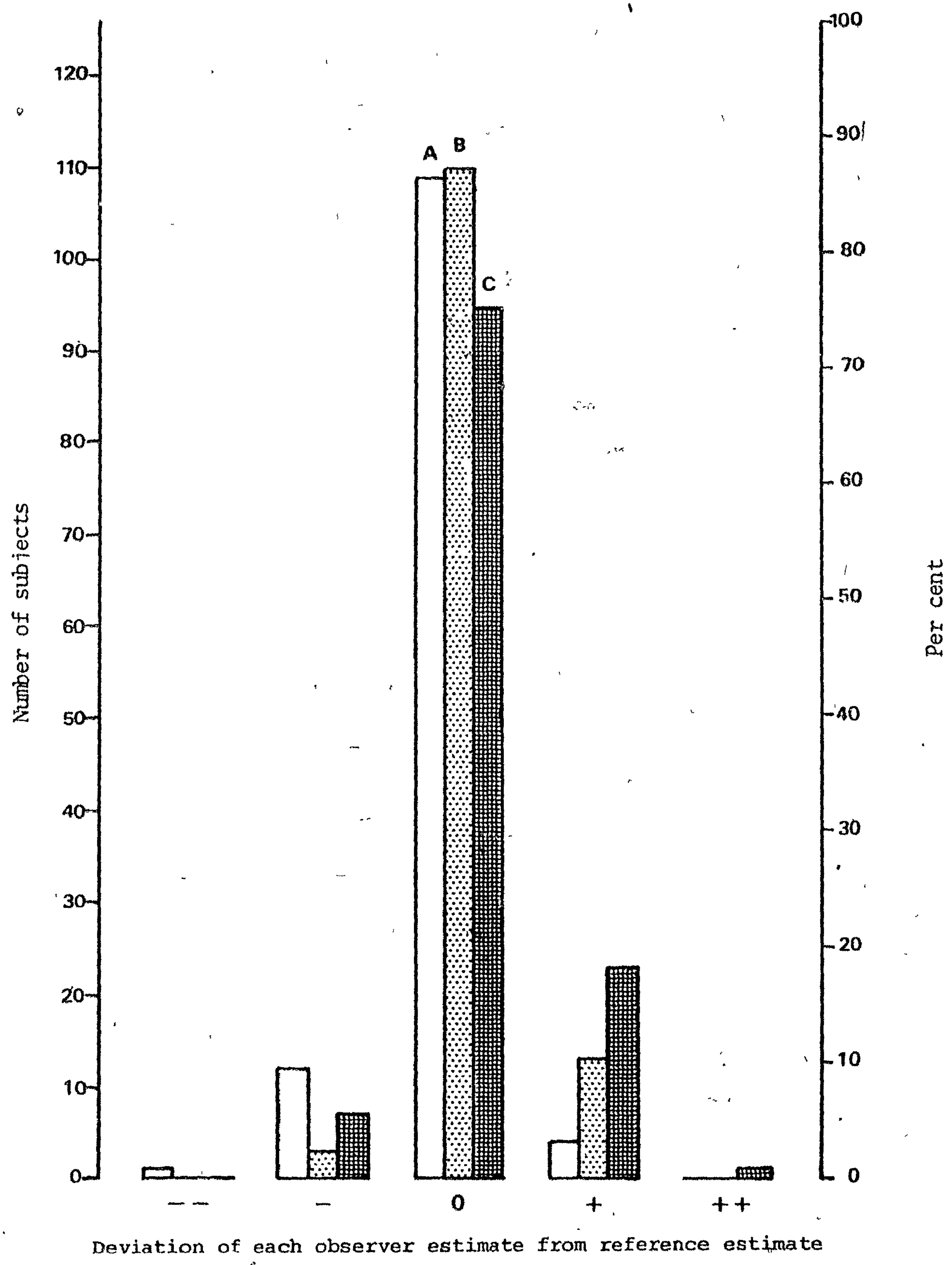


Table 11

Inter-observer Variation in Each Periodontal Parameter Estimate

		Average amount of information transmitted (binary digits)					
		According to study subjects of:					
Parameters	Observers	All 126		Initial 44		Final 44	
		amount	%*	amount	%	amount	%
Ging. stippl.	A	0.48	42.3	0.46	47.5	0.43	47.2
	B	0.67	59.0	0.43	44.4	0.54	59.0
	C	0.43	37.9	0.43	44.9	0.53	58.0
Ging. redness	A	1.15	66.5	1.15	67.1	0.98	61.3
	B	0.95	54.9	0.98	51.9	1.38	86.1
	C	0.71	41.0	0.75	43.8	0.71	44.2
Ging. bleed.	A	0.44	89.8	0.42	73.7	0.36	84.9
	B	0.40	81.6	0.48	84.2	0.42	100.0
	C	0.17	34.7	0.31	54.2	0.16	37.3
Pocket pus	A	0.21	87.5	0.20	76.1	0.16	100.0
	B	0.17	70.8	0.20	76.1	0.11	71.0
	C	0.00	0.0	0.00	0.0	0.00	0.0
Ging. recess.	A	2.21	69.7	2.31	69.2	2.65	79.6
	B	2.35	74.1	2.82	84.6	2.75	82.4
	C	1.71	53.9	2.45	73.4	2.40	72.0
Pocket depth	A	1.64	68.9	1.86	73.0	1.36	60.1
	B	1.61	67.6	2.04	80.3	1.52	67.4
	C	0.90	37.8	0.99	38.8	1.05	46.4
Epith. attach.	A	2.50	72.5	2.71	78.2	2.81	79.4
	B	2.44	70.7	2.92	84.4	2.58	73.1
	C	1.75	50.7	2.45	70.8	2.51	71.1

* Percent of information transmitted out of actual maximum information

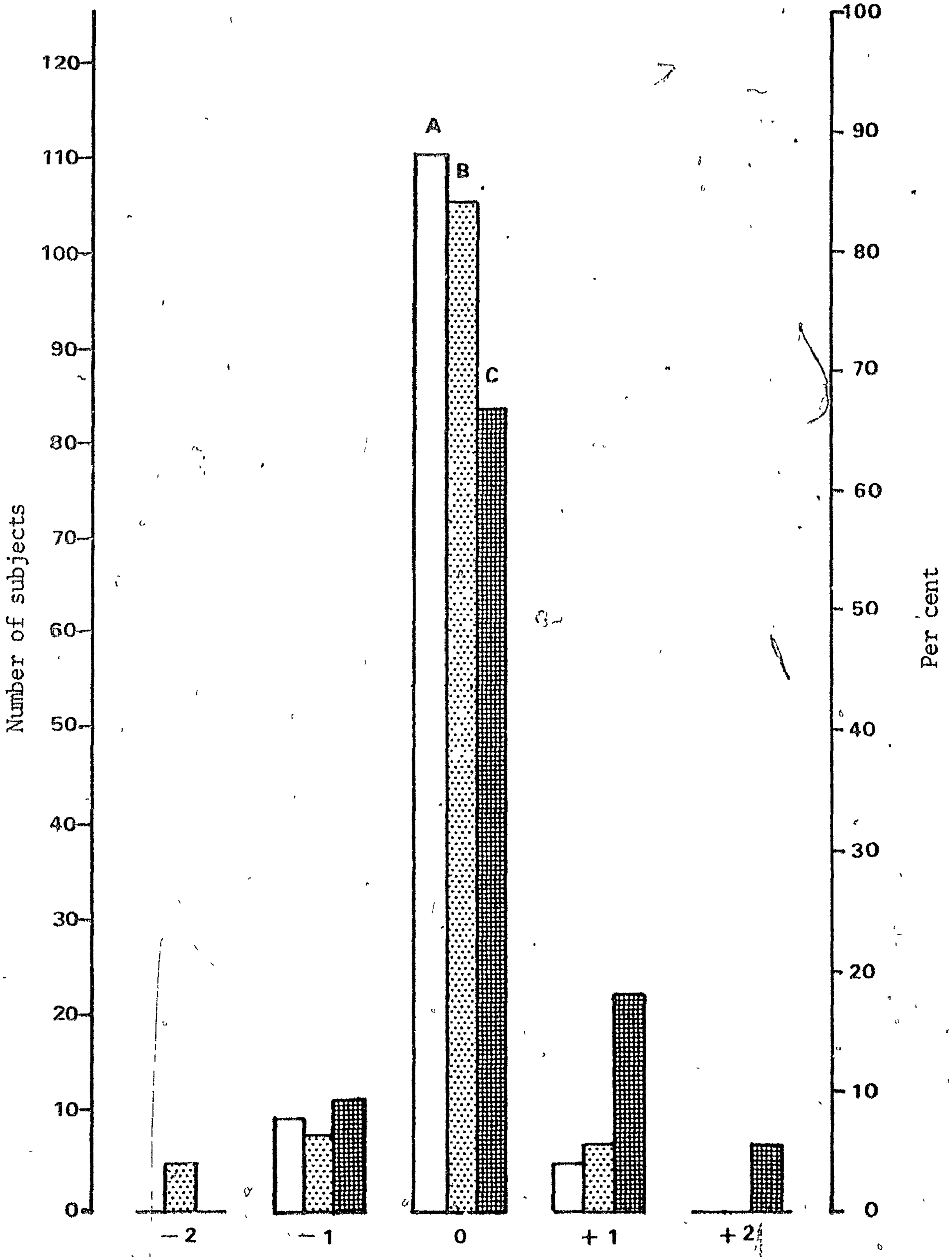
indicating that the variation was least for observer B and greatest for observer C. These values were less than one-half of the actual maximum information available. Same table also shows that there was a slight improvement in inter-observer agreement from the initial to the final gingival stippling estimate for the 44 subjects who completed the study.

4.2.2 Inter-observer variation in gingival redness and/or swelling estimate

Based on data from all 126 subjects (Fig. 5), complete agreement of observer estimates with reference estimates was quite good for observer A (88.1%) and B (84.1%). The same applied to observer C only in 66.7% of the cases. The figure also showed that while observer A and B tended to underestimate the prevalence and severity of the parameter, observer C frequently overestimated it. In some cases, gingival redness and/or swelling was scored by observer A and B as 1 while it was scored as 3 by observer C. Data from 44 subjects who completed the study also showed similar inter-observer variation in the parameter estimates. During the final examination of the 44 subjects, overestimation occurred only once in the case of observer A and B, and 12 times in the case of observer C.

The average amount of information transmitted in the gingival redness and/or swelling estimates on 126 cases were 1.15, 0.95 and 0.71 for observer A, B and C respectively, supporting the notion that observer A had the least and C the greatest variation from reference estimates as shown in Figure 5. A similar variation among three observer initial estimates on the 44

Fig. 5 Inter-observer variation of gingival redness and/or swelling estimate in 126 subjects



Deviation of each observer estimate from reference estimate subjects was observed. At the final assessment, observer B was in best agreement with reference estimates as indicated by the greatest AIT value (1.38) see Table 11.

4.2.3 Inter-observer variation in gingival bleeding estimates

Inter-observer variation of the gingival bleeding estimates is shown in Fig. 6 for the all 126 cases. In particular, observer estimates completely agreed with reference estimates in more than 90% of the cases. Estimates by observer A and B agreed with each other more frequently than with observer C. Similar inter-observer variation of this parameter estimate in initial and final assessments for 44 completed subjects was found.

Despite the relatively good agreement in three observer estimates based on deviation frequency distribution, there is great variation among them in the amount of information transmitted (Table 11). Estimates by observer A and B transmitted 73% or more of the information from the reference estimates, whereas it was 54% or less for observer C's estimates.

4.2.4 Inter-observer variation in judging the presence or absence of pus within gingival or periodontal pockets

The frequency distribution for the deviation of each observer estimate from the reference estimate in pocket pus assessment is presented in Fig. 7 for all 126 subjects. In more than 97% of the cases, estimates made by observer A and B agreed completely with the reference estimate while observer C achieved agreement in 93% or more of the cases. This was mainly because observer A and B tended to agree with each other on the presence

Fig. 6 Inter-observer variation of gingival bleeding estimate in 126 subjects

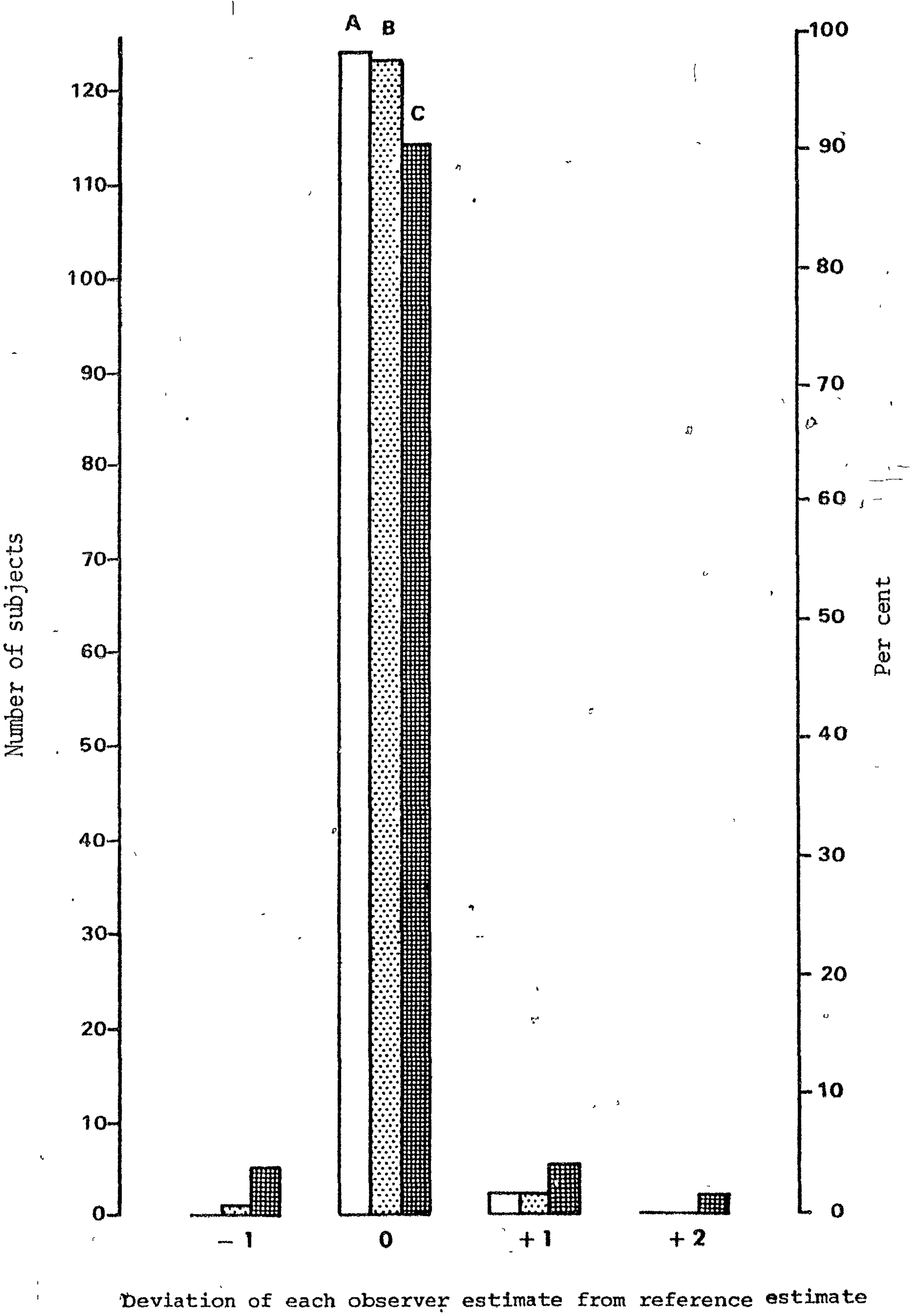
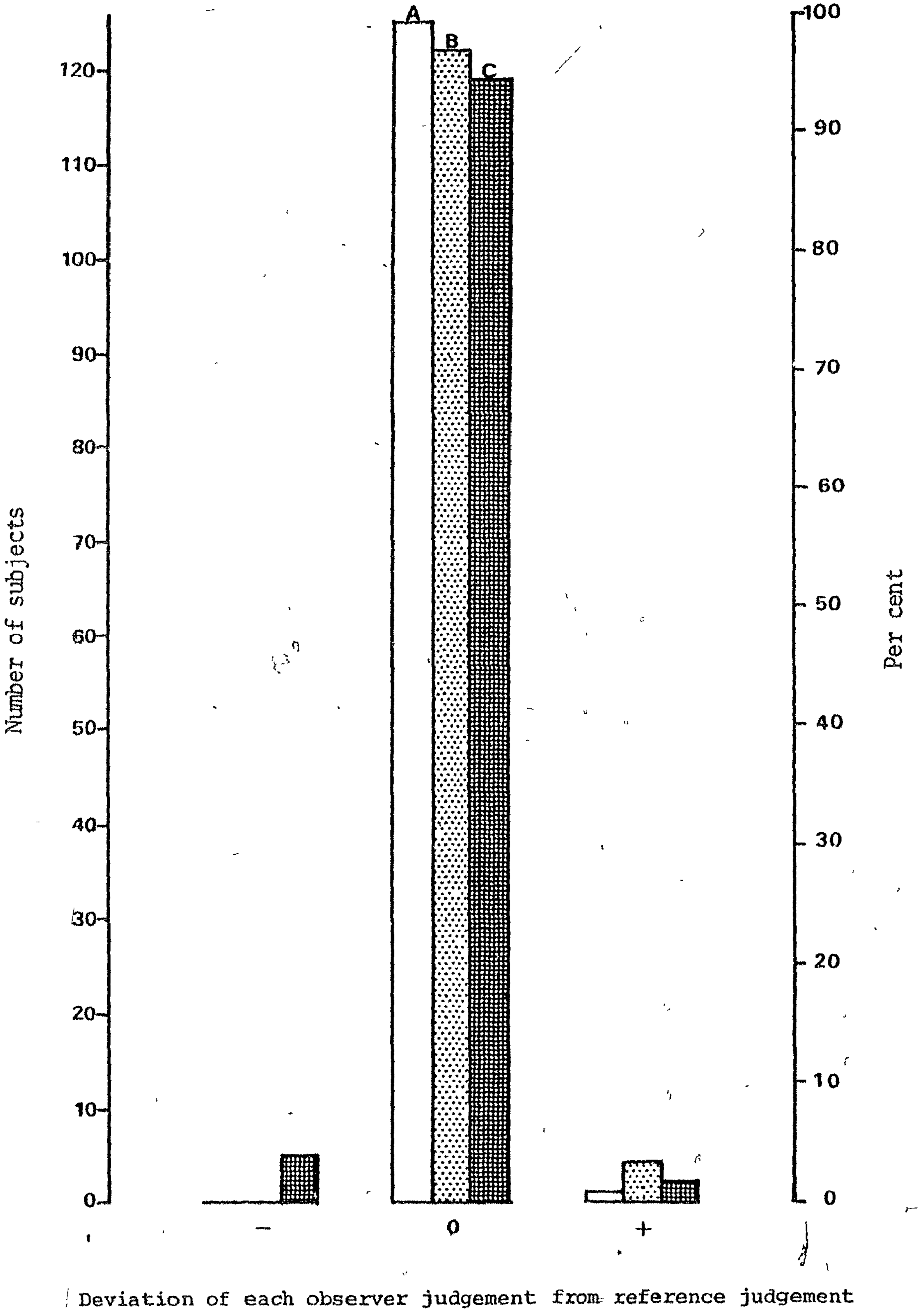


Fig. 7 Inter-observer variation in judging the presence or absence of pus in gingival or periodontal pockets in 126 subjects



of pus more frequently than did observer C. During final assessment of this parameter on 44 subjects, only in one case did observer B and C differ from the reference while observer A coincided completely with the reference estimate.

In comparison with the gingival bleeding estimate, the pocket pus estimate had much less maximum information available which was 0.26 or even less. The observer A and B's estimates transmitted 70% or more information from the reference estimates. Despite the fact that over 90% of the observer C's estimates were in complete agreement with the reference estimates, the former transmitted no information at all from the latter (see Table 11).

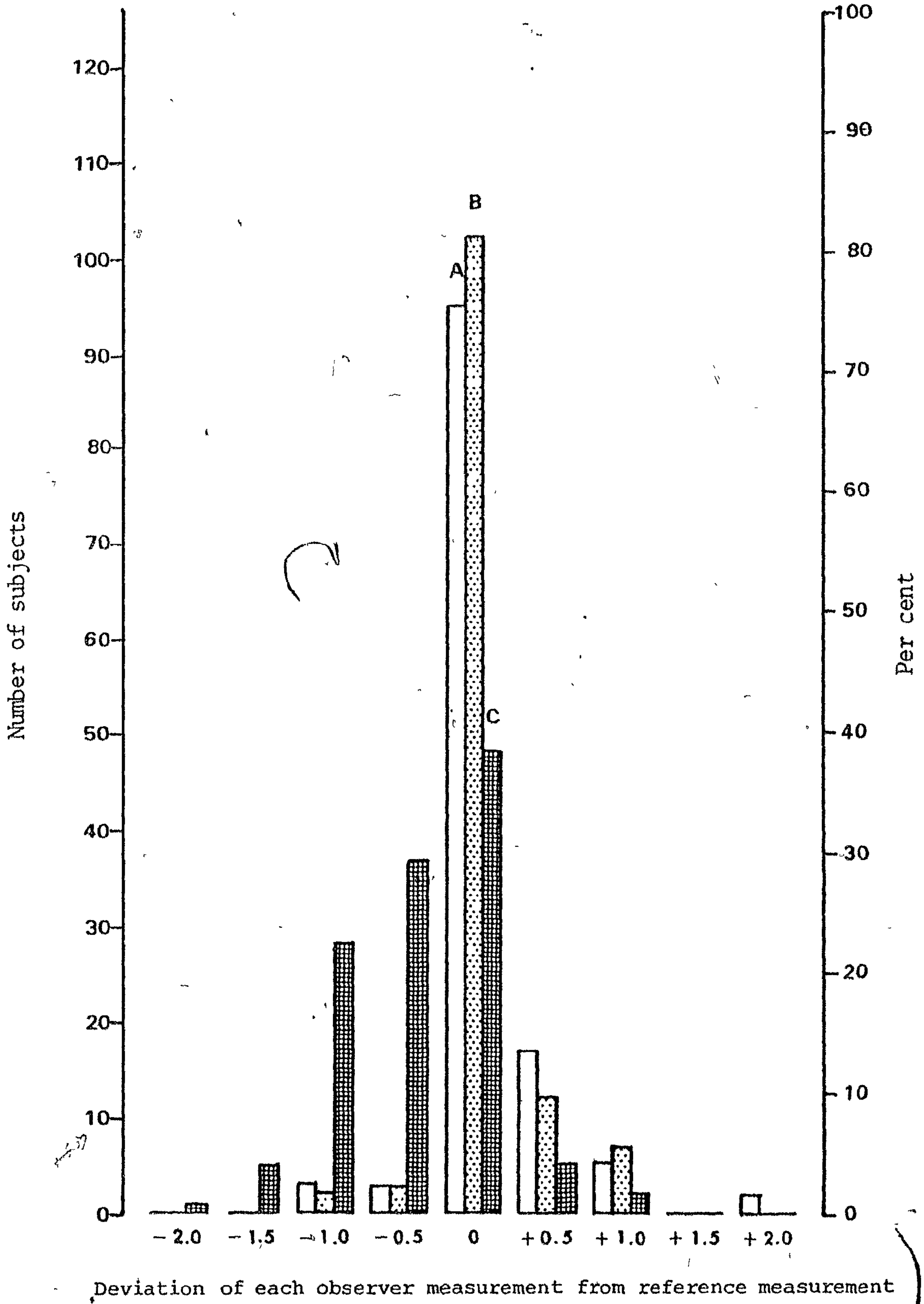
4.2.5 Inter-observer variation in gingival recession measurement

The frequency distribution of each observer measurement deviations from the reference estimate of gingival recession are shown in Fig. 8 for all 126 cases. When deviation from reference measurements was considered, nearly 80% of observer A and B's measurement were in complete agreement with the reference measurement whereas only 38% of observer C's measurements agreed with the reference. An additional 52% of cases were undermeasured by observer C by either 0.5 or 1.0 mm. Such a systematic measurement difference between observer C and observers A and B persisted at the final assessment for the 44 remaining subjects. Measurement error (variation) for the three observers ranged from 0.2 to 0.5 mm, the smallest belonging to observer B and the largest to observer C (Table 12).

In comparison with the four preceeding parameters, the actual maximum information for this parameter was much greater,

Fig. 8 Inter-observer variation of gingival recession measurement

in 126 subjects



Deviation of each observer measurement from reference measurement

Table 12

The Measurement Error (Variation) as an Estimate of Inter- and Intra-observer Variation in Measuring The Gingival Recession Level, Pocket Depth and Epithelial Attachment Level

		Measurement error (mm.)			
		Inter-observer			Intra-observer
Parameters	Observers	All 126 cases	Initial 44 cases	Final 44 cases	44 completed cases
Ging. recess.	A	0.29	0.42	0.21	0.52
	B	0.22	0.21	0.21	0.25
	C	0.47	0.42	0.54	0.47
Pocket pus	A	0.19	0.21	0.25	0.38
	B	0.16	0.15	0.23	0.34
	C	0.33	0.35	0.27	0.46
Epith. attach.	A	0.29	0.39	0.39	0.73
	B	0.28	0.28	0.35	0.46
	C	0.49	0.49	0.49	0.55

and the average amount of information transmitted in each observer measurement increases considerably (Table 11). Consequently, despite a fair amount of deviation in each observer's measurement from the reference, the information transmitted was well over 50% of the actual maximum information available, and in most cases it was 70% or greater.

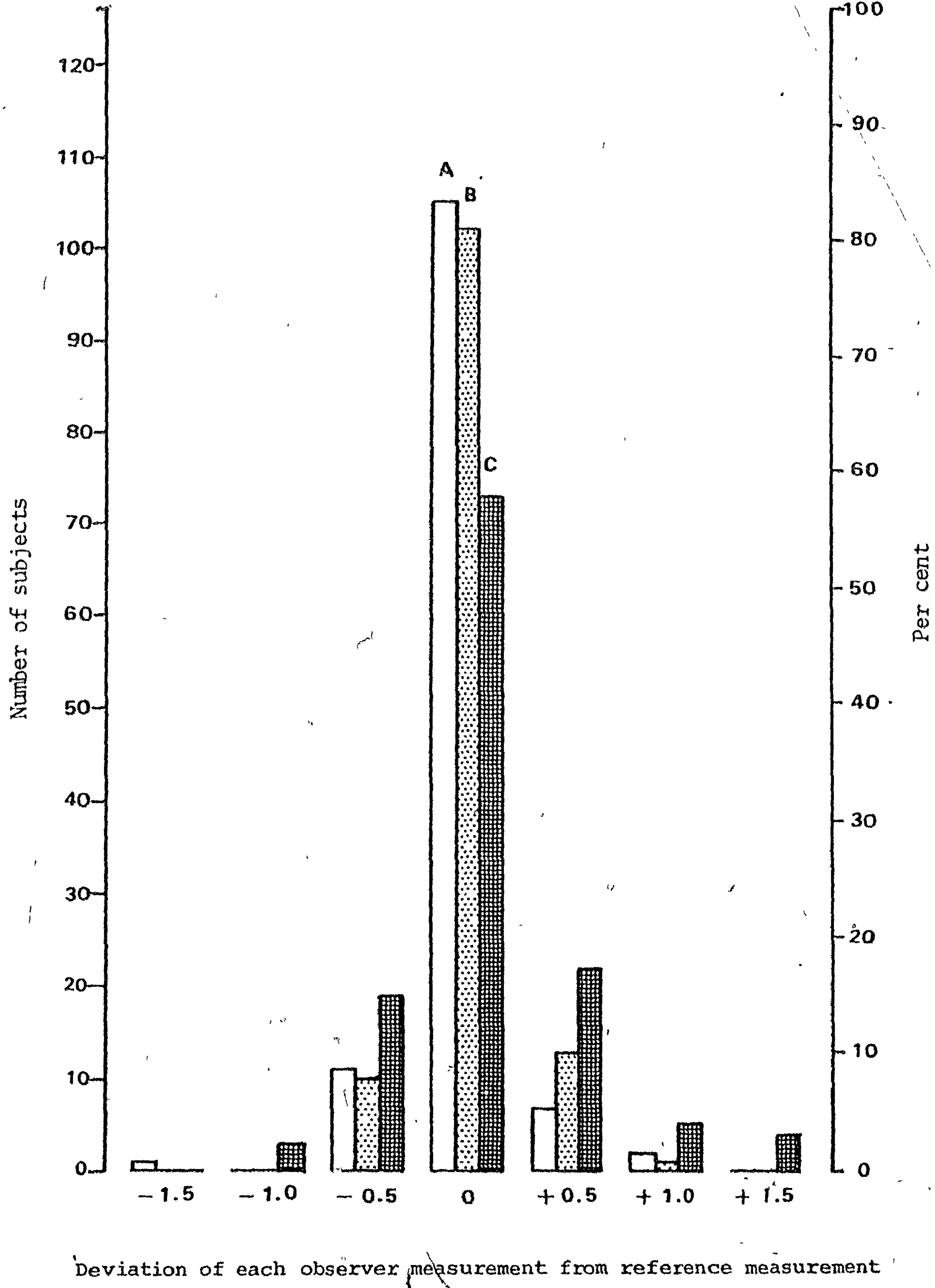
4.2.6 Inter-observer variation in gingival or periodontal pocket depth measurement

During the initial pocket depth measurement in the 126 subjects, observer A and B respectively achieved 83.3 and 81.0% agreement with the reference measurement while observer C achieved agreement in only 57.9% of the cases (see Fig 9). In situations where observer measurements differed from the reference measurement by less than ±0.5 mm, these cases were pooled together with cases of complete agreement. This resulted in 97.6%, 99.2% and 90.5% agreement for observer A, B and C respectively. The measurement error(variation)was least (0.16 mm) for observer B and greatest (0.33 mm) for observer C (Table 12).

When the 44 subjects who completed the study were considered, data from initial assessment showed inter-observer variation similar to that outlined above. At the final assessment, the degree of inter-observer variation among the three observers was quite similar. Measurement error(variation)for observer A, B and C was 0.25, 0.23 and 0.27 respectively, (see Table 12).

The actual maximum information of this parameter was much less than that of gingival recession estimate. Observer A and B's measurements transmitted over 60% of actual maximum informa-

Fig. 9 Inter-observer variation of gingival or periodontal pocket depth measurement in 126 subjects



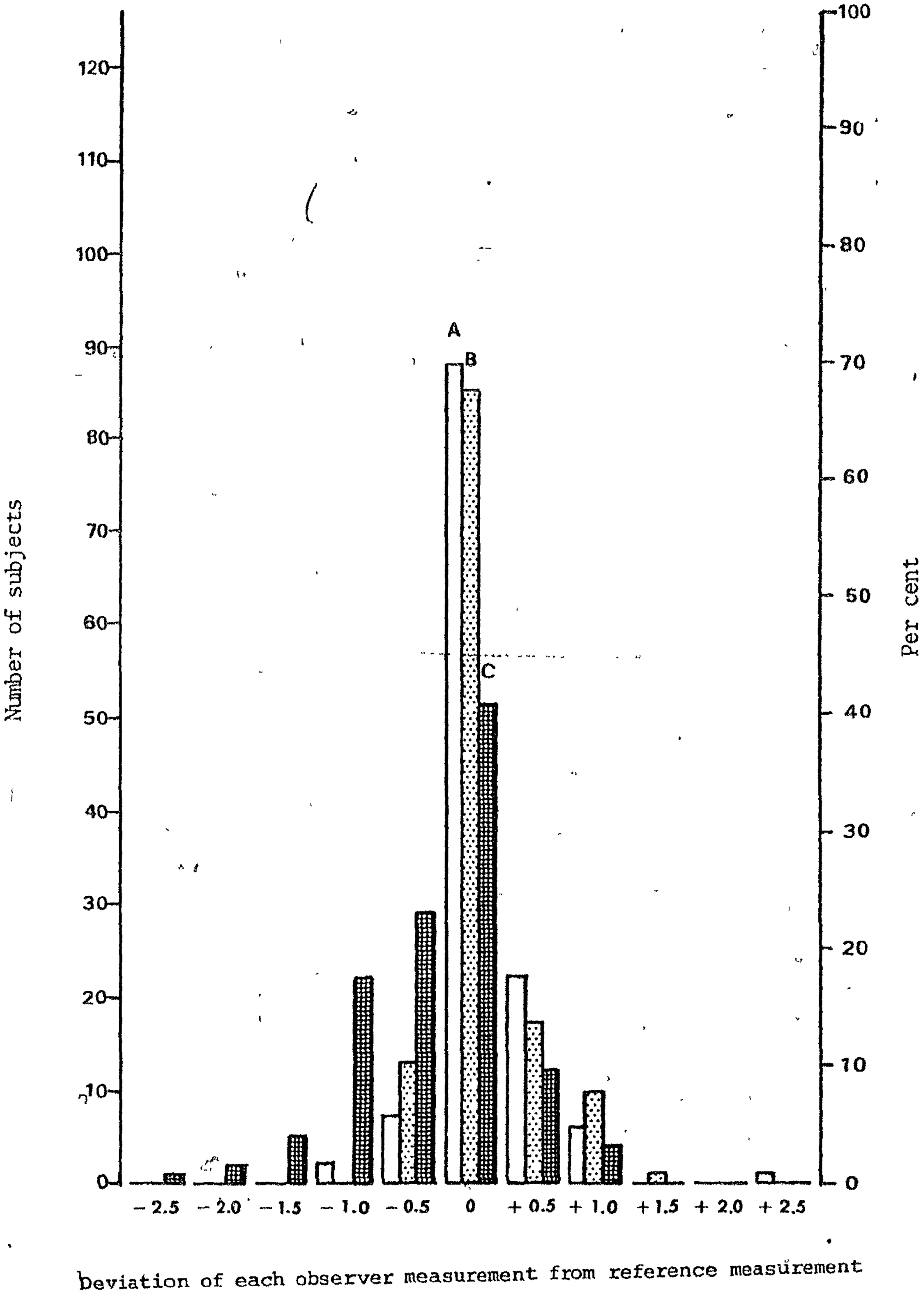
tion available, whereas AIT for observer C was less than 40% of all available information during initial assessment (see Table 11). Despite a relatively equal degree of deviation for each observer measurement from reference measurement at the final assessment on 44 remaining subjects, the information transmitted was still much less for observer C than by observer A and B.

4.2.7 Inter-observer variation in epithelial attachment level measurement

Since the epithelial attachment level was obtained by summing of the gingival recession level and the pocket depth, inter-observer variation in the epithelial attachment level measurement is a function of the variation in the two component parameters. The frequency distribution of observer measurement deviations from the reference estimate are presented in Fig. 10 for all subjects. The figure indicated that while observer C markedly underestimated the epithelial attachment level, observer B and particularly observer A, overestimated it. About 90% of the 126 cases were measured as being in the variation range of 0 to 1.0 mm by observer A and B and within -1.0 to 0.5 mm by observer C. The measurement error (variation) was 0.29, 0.28 and 0.49 mm respectively for observer A, B and C (Table 12). Such systematic inter-observer variation persisted throughout the study.

As an inverse measure of inter-observer variation, AIT supported the general impression regarding the inter-observer variation derived from the data of the deviation frequency distibution and measurement error (variation). However, it was interesting to discover that in the final assessment of 44 sub-

Fig. 10 Inter-observer variation of epithelial attachment level measurement in 126 subjects



jects, although observer C markedly underestimated the parameter whereas observer A and B only slightly overestimated it, there was only a slight difference in AIT values for the three observers.

4.2.8 Summary of the inter-observer variation in parameter estimate or measurement

The nature of the inter-observer variation for each parameter assessed suggests that there were three major groups of parameters each with rather distinct characteristics with regard to the inter-observer variation in the estimates. In the first group, gingival stippling and gingival redness, complete agreement of each observer's estimate with reference estimate was greater than 80% for observer A and B and about 70% for observer C. The average amount of information transmitted was moderate-- about 0.5 for gingival stippling and 1.0 for gingival redness. Respectively, this accounted for nearly 50% and 60% of the actual maximum information in each parameter.

In the second group were gingival bleeding and pocket pus. Since only a few cases demonstrated positive signs, more than 90% of the observer estimates were in agreement with reference estimates. In the cases where gingival bleeding or pocket pus existed, estimates among the three observers varied considerably, especially observer C against observers A and B. Whereas observer A and B's estimates sometimes transmitted nearly 100% of actual maximum information, observer C's estimate transmitted practically no information despite the fact that there was over 90% agreement among the three observers.

The last group contained gingival recession level, pocket depth and epithelial attachment level. Both the amount of information transmitted and the percentage of actual maximum information rose considerably. The inter-observer variation was significantly reduced. In many cases in which observer A and B recorded 0 mm of gingival recession, observer C measured 0.5 to 1.0 mm less gingival recession. In comparison to observers A and B, observer C slightly overmeasured the pocket depth. Consequently, for the parameter of epithelial attachment level, observer A and B slightly overmeasured (0.5 to 1.0 mm) and observer C moderately undermeasured it (-0.5 to -1.0 mm). The measurement error or variation in epithelial attachment level was approximately 0.4 mm, 0.3 mm and 0.5 mm for observer A, B and C respectively.

4.3 Intra-observer variation of each parameter estimate or measurement

Although each subject was asked to return for a re-assessment on the 7th day following the initial examination, the interval between two assessments varied greatly from one subject to another. Of the 44 persons who completed the study and were consequently available for intra-observer variation assessment, only 18 returned on the 7th day. An additional 9 persons came back between the 4th and the 6th day, while 12 and 4 subjects returning during the second and third week respectively. One person came on the 27th day after initial assessment. It is reasonable to assume that some, however slight, changes in parameter severity may have occurred between the two examinations, especially those involving gingival tissue only. Therefore, the estimate of intra-observer variation in this study actually includes, in addition to real intra-observer variation, some possible severity changes taking place between the two assessments.

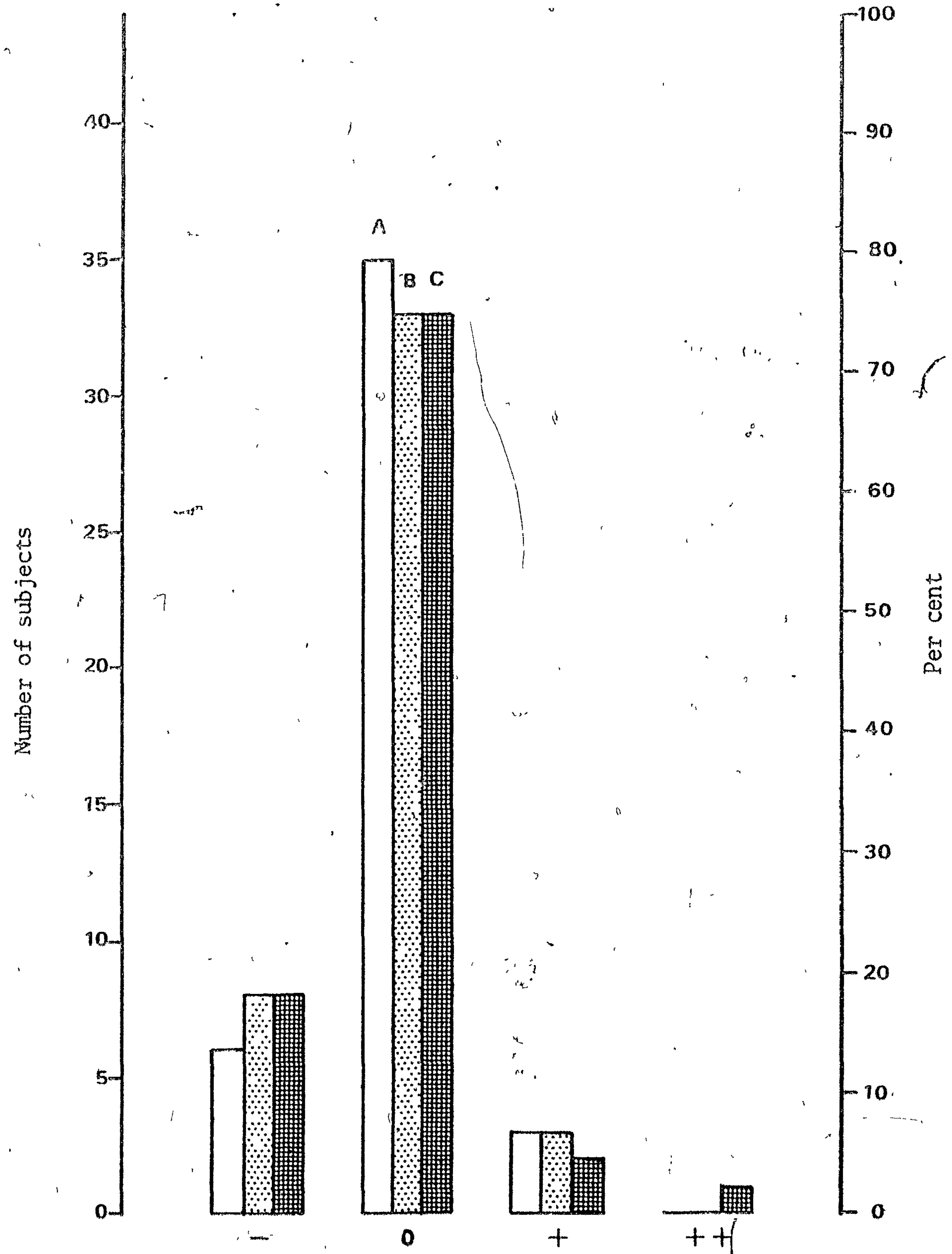
4.3.1 Intra-observer variation in gingival stippling estimate

The frequency distribution of the deviations between the first and the second gingival stippling estimates is shown in Fig. 11. In 33 or more cases, each observer made the same estimate for the first and second scoring. During the second assessment, all observers estimated lower scores than they did initially. Based on the deviation distribution, observer A seemed to achieve the best scoring reproducibility. When the statistics of the average amount of information are considered, data from observer A actually transmitted the least amount of information, therefore, indicating the greatest within observer variation (see Table 13). Data from observer B and C are quite comparable for both deviation distribution and average amount of information transmitted (AIT).

Comparing AIT for inter- and intra-observer variation (Table 11 and 13), the data indicate that both observer B and C are given to similar inter and intra-observer variation in gingival stippling estimate. Observer A, however, achieved better agreement with observers B and C than he did with himself.

4.3.2 Intra-observer variation in gingival redness and/or swelling estimate

Fig. 11 Intra-observer variation of gingival stippling estimate in 44 subjects who remained in the study



Deviation of the second from the first estimate

Table 13

Intra-observer Variation of Each Parameter Assessment in 44 Subjects Who Remained in The Study

		Average amount of information transmitted (binary digits)		
		Observers		
Parameters		A	B	C
Ging. stippling	amount	0.23	0.44	0.42
	percent*	25.6	36.1	32.3
Ging. redness	amount	0.75	0.87	0.82
	percent	44.6	56.9	43.7
Ging. bleeding	amount	0.10	0.27	0.16
	percent	23.8	36.2	51.6
Pocket pus	amount	0.02	0.20	0.00
	percent	5.6	56.0	0.0
Ging. recession	amount	1.65	2.44	2.15
	percent	53.2	73.7	62.3
Pocket depth	amount	0.95	1.15	0.68
	percent	37.6	43.1	28.9
Epith. attachment	amount	2.14	2.46	2.41
	percent	61.6	66.9	70.1

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* Percent of information transmitted out of actual maximum information

The intra-observer variation in gingival redness and/or swelling estimates by each observer is presented in Fig. 12. Observers A, B and C achieved complete reproducibility in 34 (77.3%), 36 (81.8%) and 31 (70.5%) of the 44 completing subjects. Practically all the intra-observer estimate differences in the remaining subjects were the result of under or over-scoring by one unit.

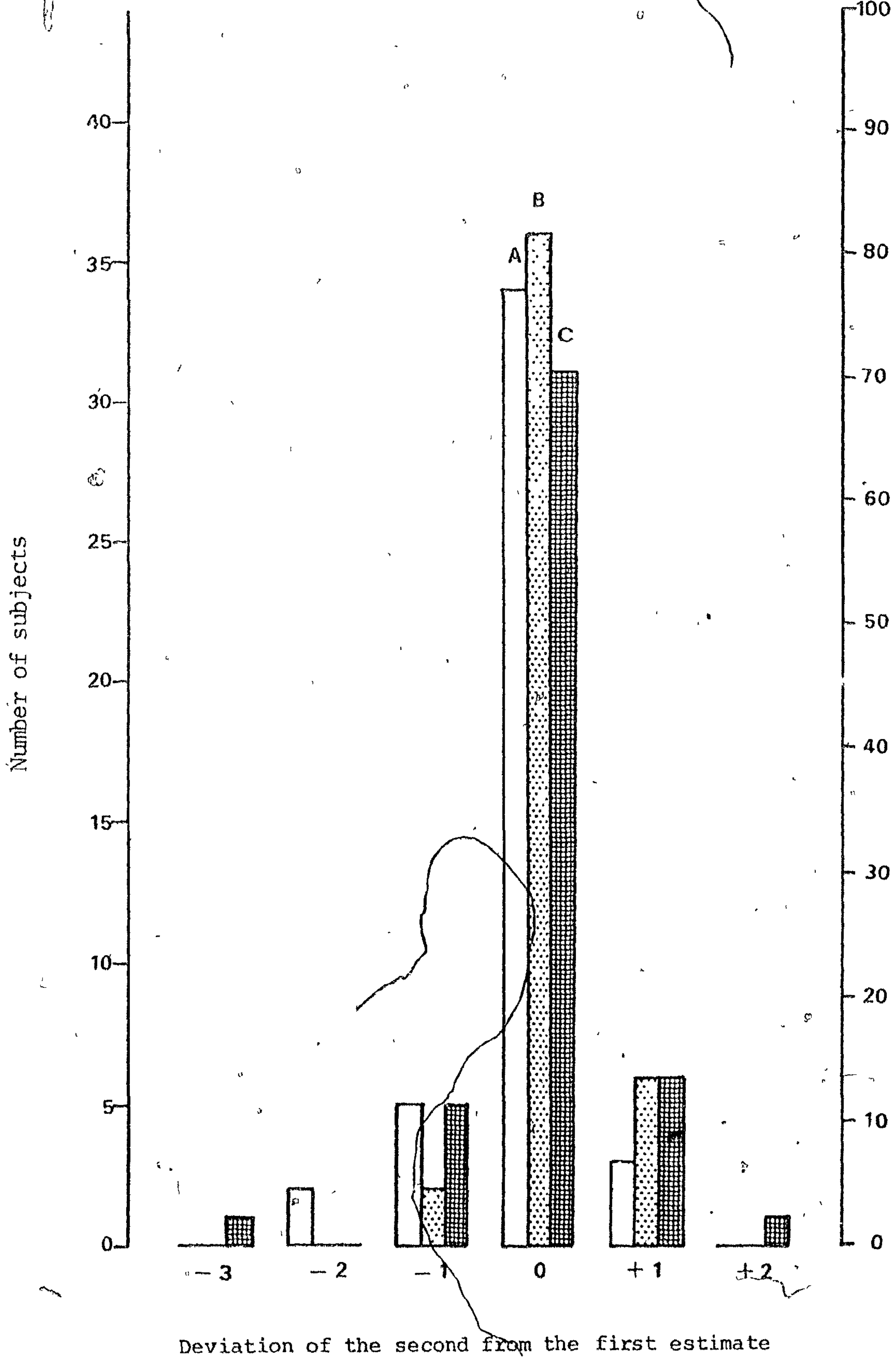
The statistics of the average amount of information transmitted (AIT) were 0.75, 0.87 and 0.82 for observer A, B and C respectively (see Table 13), indicating that intra-observer variation was least for observer B and greatest for observer A. Comparing these data with the AIT in Table 11, it becomes clear that while observers A and B agreed well with each other and to a lesser extent with observer C, yet in terms of reproducibility, each observer achieved relatively the same degree of intra-observer variation.

4.3.3 Intra-observer variation in gingival bleeding estimate

The reproducibility in gingival bleeding assessment was achieved in 38 subjects (86.4%) by observers A and B and in 42 subjects (95.5%) by observer C (Fig. 13). Practically all the remaining subjects were over- or under-scored by one unit only in successive assessments.

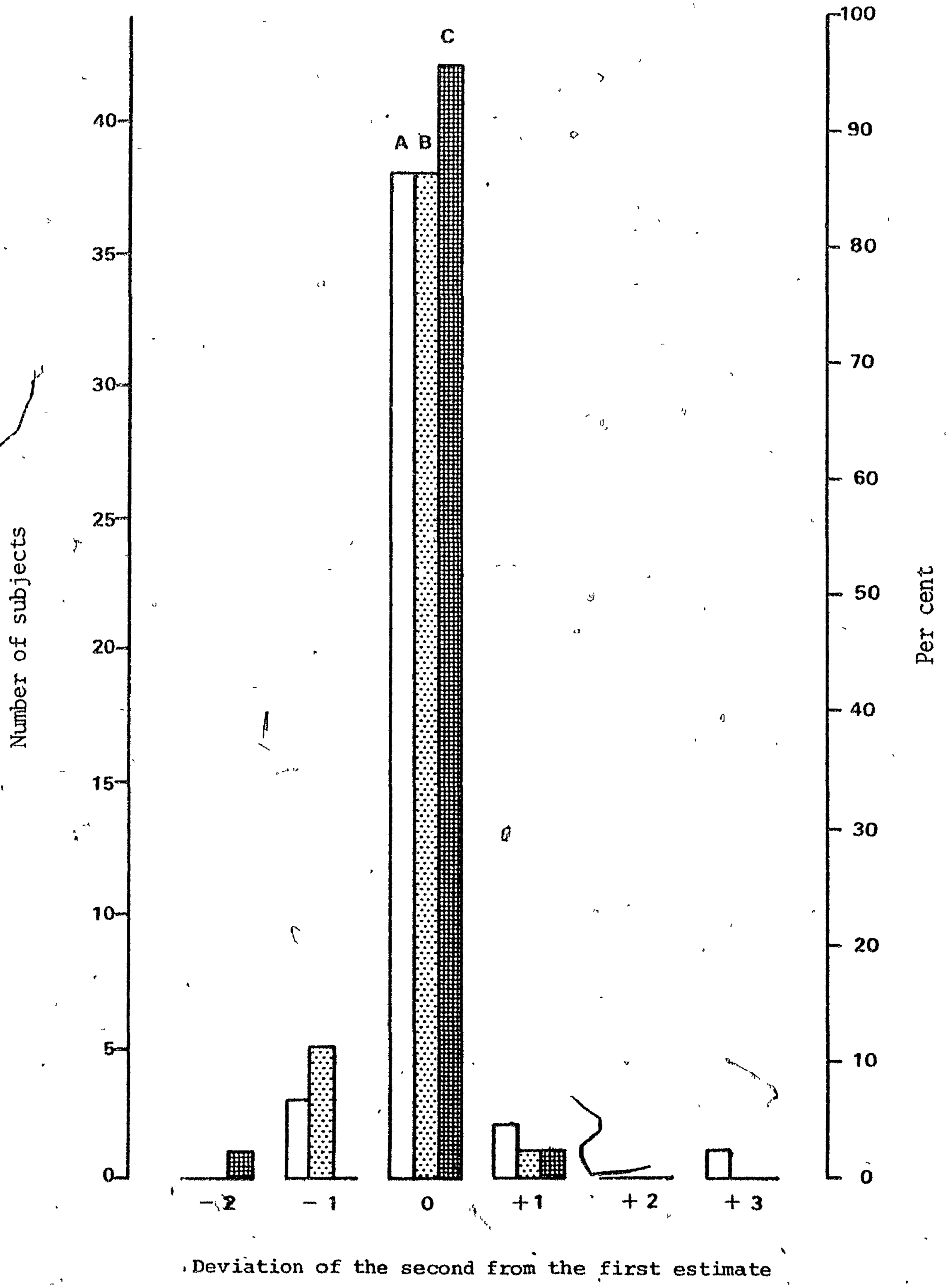
For the gingival bleeding estimate, the average amount of information transmitted was 0.10, 0.27 and 0.16 for observers A, B and C respectively (Table 13). This indicates that the intra-observer variation was least for observer B and greatest for observer A.

Fig. 12 Intra-observer variation of gingival redness and/or swelling estimate in 44 subjects who remained in the study



Deviation of the second from the first estimate

Fig. 13 Intra-observer variation of gingival bleeding estimate in 44 subjects who remained in the study



Comparing these results with those of inter-observer variation (Table 11), the data indicate that observer B agreed well not only with observers A and C, but also with himself, observer A achieved better agreement with observer B and C than he did with himself, and that observer C achieved better agreement with himself than he did with observers A and B.

4.3.4 Intra-observer variation in judging the presence or absence of pus in gingival or periodontal pockets

In successive assessments of the presence or absence of pus in gingival or periodontal pockets, observers B and C recorded the same estimates in 43 cases. Observer A obtained identical scores for 40 subjects (Fig. 14).

Despite such good reproducibility, the average amount of information transmitted was only 0.02, 0.20 and 0.00 for observers A, B and C respectively (Table 13). Thus, the application of the statistic of AIT as an inverse measure of observer variation was of limited use. This was true in the case of observer A and particularly so for observer C.

Based on inter-observer variation (Fig. 7) and intra-observer variation (Fig. 14), observer C's estimates corresponded poorly to those of observer A and B. Observer A achieved **least** reproducibility in successive assessments.

4.3.5 Intra-observer variation in gingival recession measurement

The frequency distribution of the deviations in repeated assessments of gingival recession is shown in Fig. 15. There were 19 (43.2%), 28 (63.6%) and 14 (31.8%) subjects for whom observers

Fig. 14 Intra-observer variation in judging the presence or absence of pus in gingival or periodontal pockets of 44 subjects who remained in the study

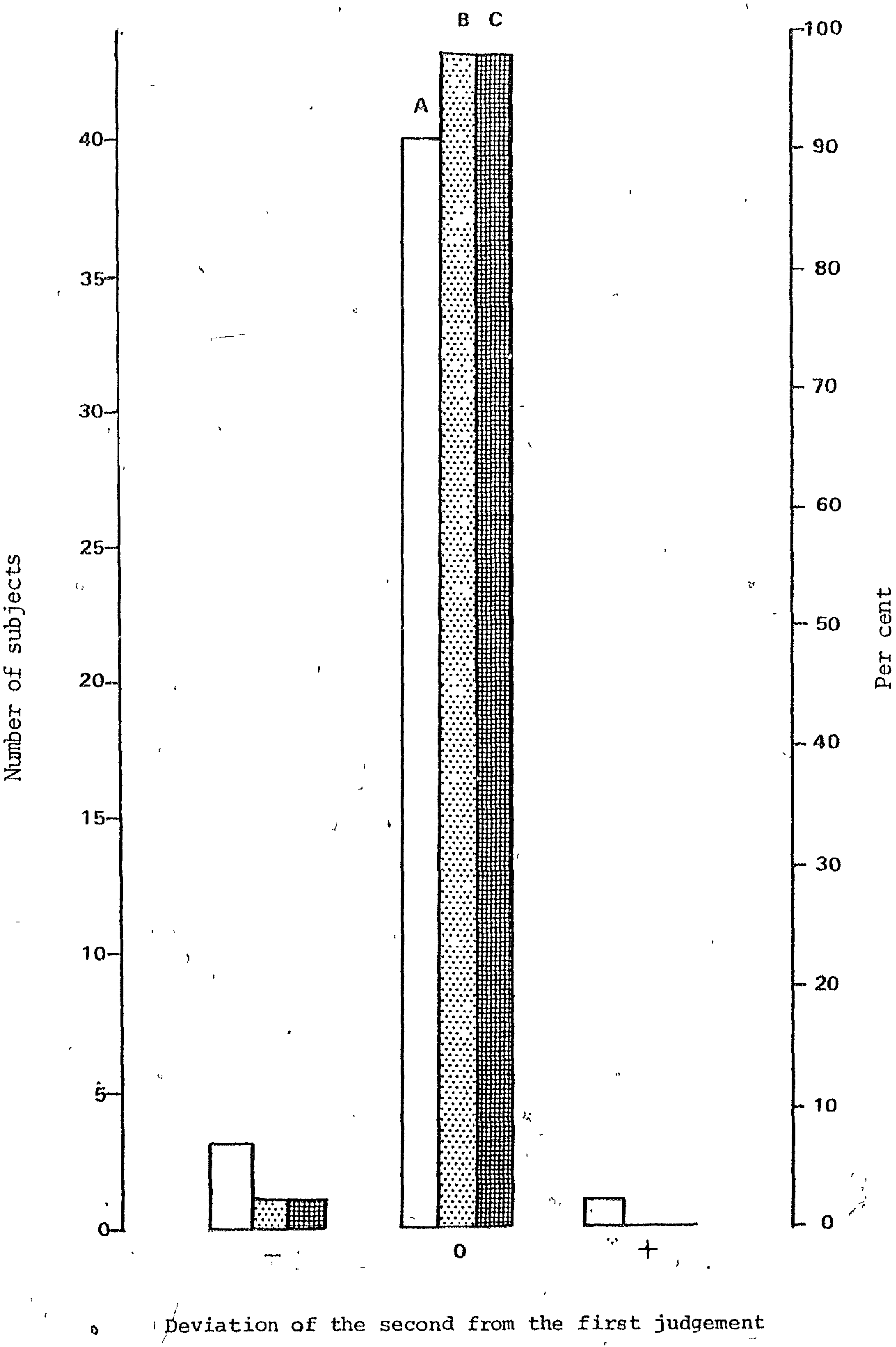
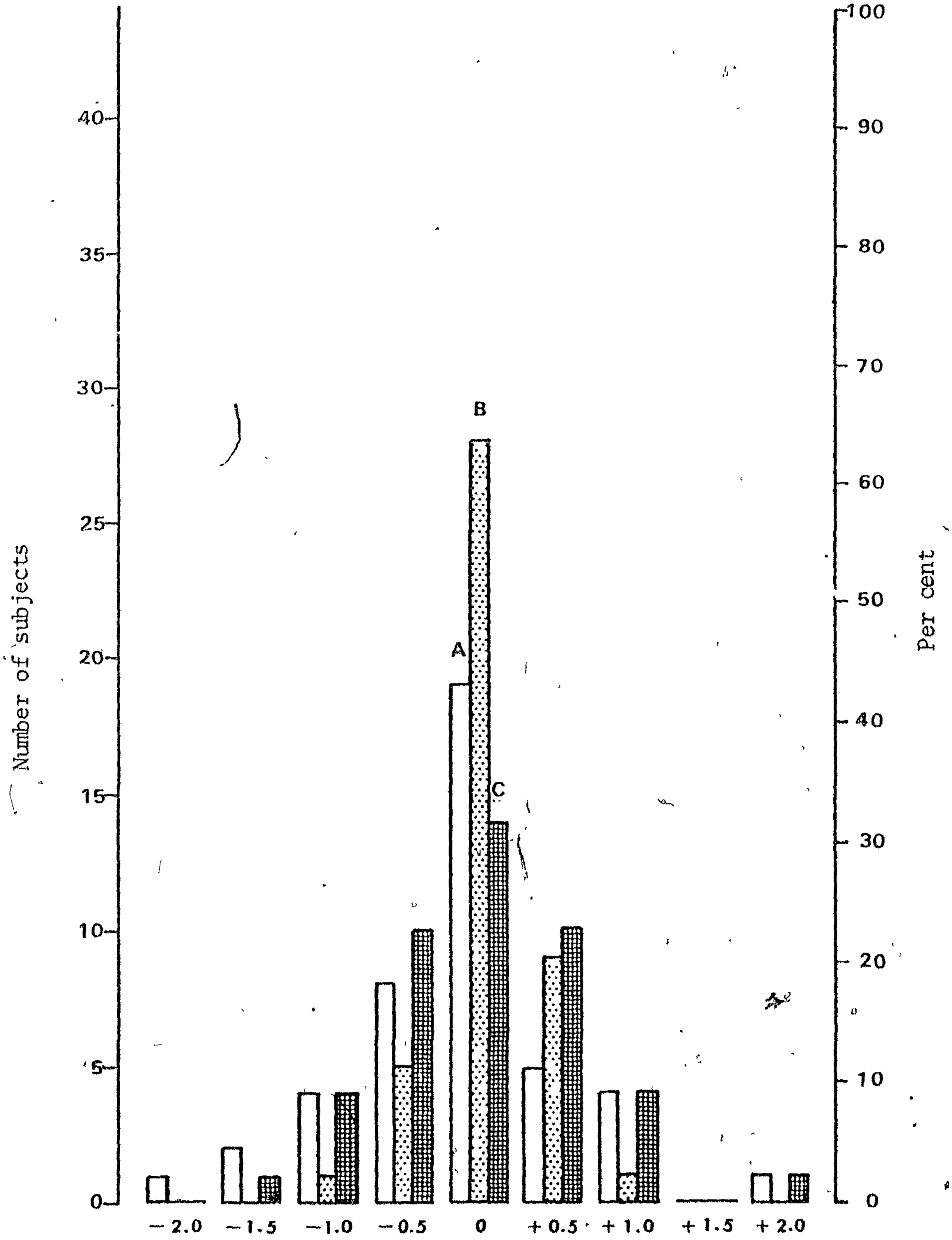


Fig. 15 Intra-observer variation of gingival recession measurement in 44 subjects who remained in the study



Deviation of the second from the first measurement

A, B and C respectively reproduced the same measurements. If one considers all deviations of ±0.5 mm as being negligible, then observers A, B and C achieved reproducibility for 32 subjects (72.8%), 42 subjects (95.5%) and 34 subjects (77.3%) respectively. The maximum difference in repeat assessments was ±2.0 mm.

Measurement error (variation) provides a precise data for each observer's reproducibility in making the measurement. It was 0.52, 0.25 and 0.47 mm for observer A, B and C respectively, see Table 12. Thus, observer B was considerably more consistent in assessing gingival recession.

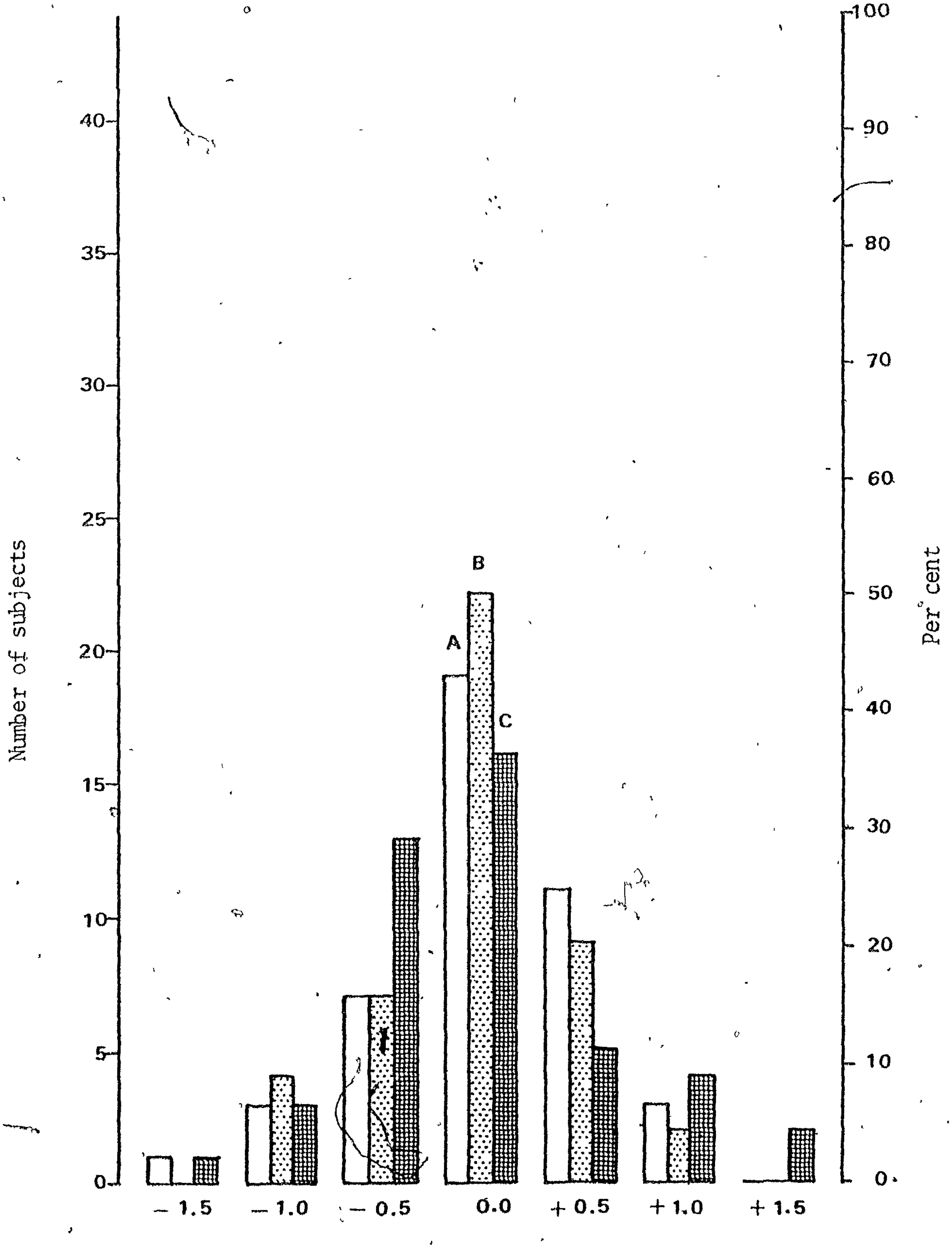
The average amount of information transmitted for observers, A, B and C was 1.65, 2.44 and 2.15 respectively (Table 13). These data substantiate the conclusion drawn from both deviation frequency distribution and measurement error.

Judging from both AIT and measurement error, there is some indication that measurement variation within observers was greater than that between observers, see Table 11, 12 and 13. This is particularly obvious for observer A and less so for observers B and C.

4.3.6 Intra-observer variation in gingival or periodontal pocket depth measurement

The frequency distribution of the variation in repeated measurements of gingival or periodontal pocket depth is shown in Fig. 16. Complete reproducibility occurred in 19 cases (43.2%), 22 cases (50%) and 16 cases (36.4%) for observer A, B and C respectively. If deviation of ±0.5 mm were considered as being negli-

Fig. 16 Intra-observer variation of gingival or periodontal pocket depth measurement in 44 subjects who remained in the study



Deviation of the second from the first measurement gible, reproducibility was achieved in 37, 38 and 34 subjects respectively for the same three observers. The maximum deviation was ±1.5 mm.

The observer B achieved the lowest measurement error (0.34 mm) and was followed by observer A (0.38 mm) and C (0.46 mm), see Table 12. The statistics of AIT were 0.95, 1.15 and 0.68 respectively for observers A, B and C (Table 13). Thus, all data presented hitherto indicate that observer B was able to achieve the best reproducibility in the measurement of pocket depth.

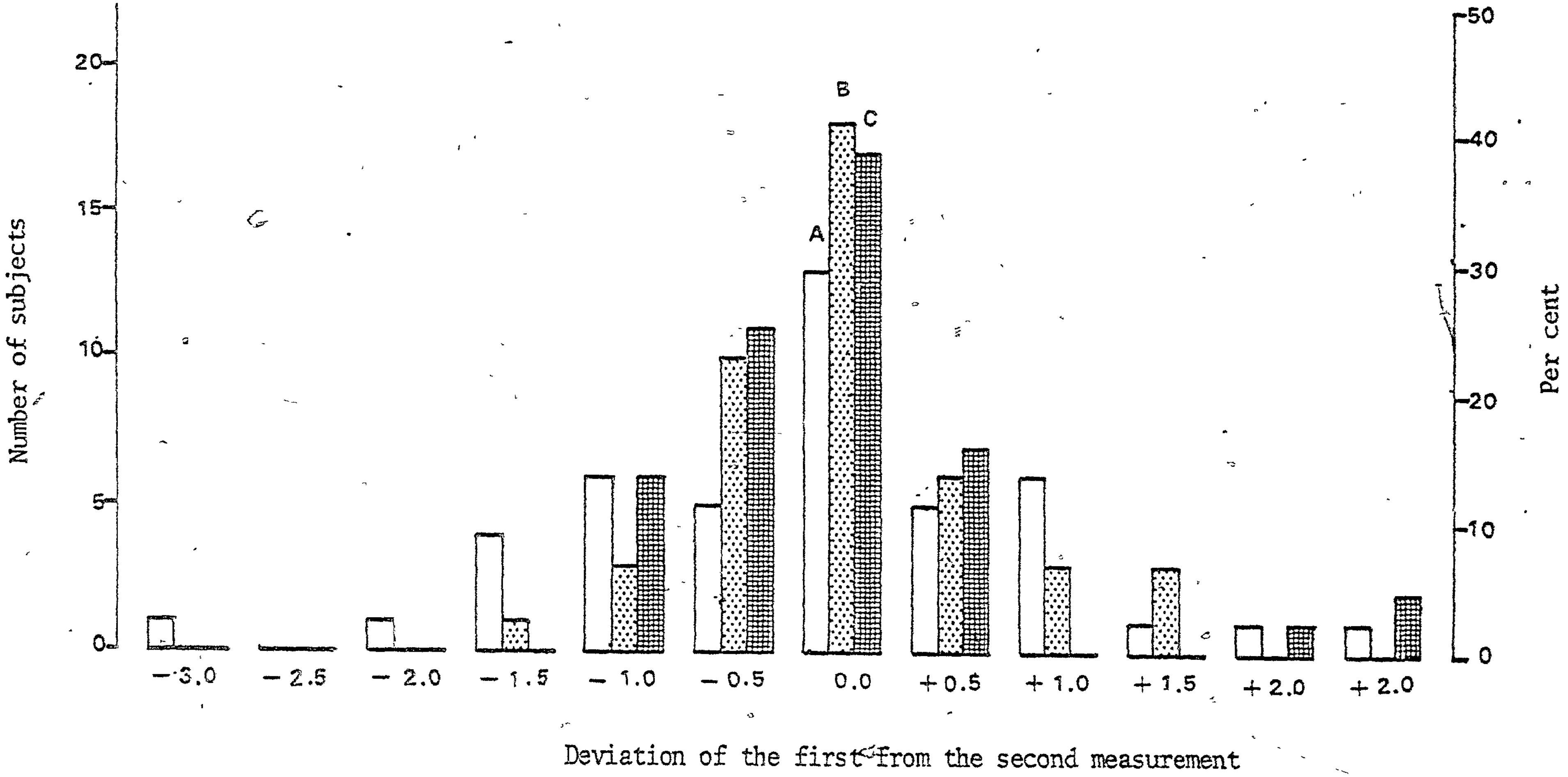
Considering all observers together, intra-observer variation for this parameter is considerably greater than inter-observer variation, see Table 11, 12 and 13.

4.3.7 Intra-observer variation in epithelial attachment level measurement

The frequency distribution of within observer variation in repeated assessments of epithelial attachment level is presented in Fig. 17. Complete reproducibility in measurements was achieved for 13 (29.5%), 18 (40.9%) and 17 (38.6%) by observers A, B and C respectively. Adding the cases with a deviation equal to or less than ±0.5 mm in repeated measurements, reproducibility increases to 24 (54.6%), 34 (77.3%) and 35 (79.5%) subjects respectively. In more than 90% of the cases measured by observer B and C and 80% by observer A, the differences between repeated measurements are 1 mm or less.

The measurement errors by observers A, B and C were 0.73 mm, 0.46 mm and 0.55 mm respectively (Table 12). These values

Fig. 17 Intra-observer variation in repeat measurements of the epithelial attachment level in 44 subjects



were greater than inter-observer measurement errors.

The average amount of information transmitted from repeated epithelium attachment level measurements was 2.14, 2.46 and 2.41 for observor A, B and C respectively (Table 13). The statistics of both AIT and measurement error indicate a better intra-observer agreement by observer B and C measurements than by observer A.

4.3.8 Summary of intra-observer variation in parameter estimate or measurement

In obtaining successive measurements of gingival stippling and gingival redness, each observer achieved complete reproducibility in 75% and 70% of the subjects assessed. The statistic of the average amount of information transmitted suggests that within observer variation was greater for observer A than for observer B and C.

Because of the few cases which demonstrated gingival bleeding or pus within the gingival or periodontal pocket, in over 90% of the cases the three observers achieved complete reproducibility in estimates or judgements. The statistic of AIT is however, of little use in the case of the gingival bleeding estimate and of no use in the case of pocket pus. Here, AIT fails as an inverse measure of intra-observer variation.

For repeated assessments of gingival recession and pocket depth, intra-observer measurement differences of 0.5 mm or less were achieved by all observers in more than 70% and 77% of cases respectively. The measurement error for gingival recession assessment varied from 0.25 mm for observer B to 0.52 mm for observer A. The corresponding variation in pocket depth assessment varied from 0.34 mm for observer B to 0.46 mm for observer C, and in epithelial detachment from 0.46 mm for observer B to 0.73 mm for observer A.

For all parameters, both the statistics of measurement error and average information transmitted suggest that observer B's estimates and measurements had the least intra- and inter-observer variation. Observer C's estimates and measurements had less intra-observer but greater inter-observer variation than had observer A. It is also apparent that intra-observer variation was greater than inter-observer variation for all parameters assessed.

4.4 The association in each selected pair of parameters

Since the periodontal index under discussion is a function of the component parameters, its construction is strongly dependent on the interrelationship among parameters. Gingival redness and each of the other parameters except gingival or periodontal pocket pus were considered in pairs for preliminary testing of the parameters' interrelationship. In addition, the relationship between the gingival recession level and pocket depth was also studied. Both observer C and median initial estimates or measurements were used for this purpose.

The average amount of information transmitted by each parameter pair is shown in Table 14. Of the 1.73 of actual maximum information in the median gingival redness estimate, only 0.29, 0.08, 0.45, 0.20 and 0.54 are associated with gingival stippling, gingival bleeding, gingival recession, pocket depth

Table 14

The Relationship Within Selected Pairs of Parameters: The AIT Estimation Based On Original Severity Classification

Parameter pair	Number of severity categories	Average amount of information transmitted *	
		Source of data	
		Median	Observer C
Ging. stippl. vs ging. red.	3, 4	0.29	0.40
Ging. bleed. vs ging. red.	4, 4	0.08	0.09
Ging. recess. vs ging. red.	19, 4	0.45	0.58
Pocket depth vs ging. red.	10, 4	0.20	0.29
Epith. attach. vs gin. red.	20, 4	0.54	0.63
Pocket depth vs ging. recess.	10,19	0.59	0.78

* The actual maximum information in gingival redness estimate is 1.73 and 1.89 respectively for median and observer C data.

and epithelial detachment estimates respectively. Despite the fact that these values are relatively small in proportion to the total information for gingival redness, Chi square tests revealed that except for gingival bleeding estimates, the two-way distribution of gingival redness estimate with each of gingival stippling, gingival recession, pocket depth and epithelial detachment level was far from being a random distribution. Data from observer C estimates showed slightly closer association in each parameter pair than did those from the median estimates (Table 14).

The average amount of information transmitted by each parameter with regard to gingival redness is to a great extent influenced by its number of severity categories -- for a constant relationship, the more classifications a parameter has, the more information it will transmit. This phenomenon is well demonstrated in Table 14. Except for gingival stippling, the amount of AIT is proportionally associated with the number of severity classifications in each parameter. It is, therefore, not totally justified to compare the AIT between pairs in order to determine the relative strength of the association. For this reason, measurements of gingival recession level, pocket depth and epithelial detachment level, which had greater than 4 severity categories, were each re-grouped into four groups according to severity gradients (see footnote of Table 15). The cutting points between groups were chosen so that the final frequency distribution would not be highly skewed in one direction. In addition, groupings were chosen to be compatible with the general clinical

Table 15

The Relationship Within Selected Pairs of Parameters: The AIT Estimation Based on An Equal Number of Severity Classification *

Parameter pair	Number of severity categories	Average amount of information transmitted **	
		Source of data	
		Median	Observer C
Ging. stippl. vs ging. red.	3, 4	0.29	0.40
Ging. bleed. vs ging. red.	4, 4	0.08	0.09
Ging. recess. vs ging. red.	4, 4	0.26	0.45
Pocket depth vs ging. red.	4, 4	0.15	0.16
Epith. attach. vs ging. red.	4, 4	0.21	0.40
Pocket depth vs ging. recess.	4, 4	0.18	0.06

* Gingival stippling has only 3 severity categories. The estimates of gingival recession, pocket depth and epithelial attachment level were re-grouped into 4 categories as follow:

Parameter	Group 1	Group 2	Group 3	Group 4
Ging. recess. (mm)	<0	0.0-1.0	1.5-3.0	3.5+
Pocket depth (mm)	0.0-0.5	1.0-1.5	2.0-2.5	3.0+
Epith. attach. (mm)	≤ 0	0.5-1.0	1.5-2.0	2.5+

** Actual maximum information in gingival redness estimate is 1.73 and 1.89 respectively for median and observer C data.

concept of normal, mild, moderate and severe stages for each parameter. Table 15 presents the results when re-grouped data of equal classifications were used for AIT estimation. In contrast to the relatively greater amount of information transmitted by gingival recession and epithelial detachment than by gingival stippling when the original classification was used (Table 14), the information transmitted by the first two parameters was similar to or less than that by the third parameter when equal classifications were employed. Thus, the gingival redness estimate is about equally associated with gingival stippling, gingival recession and epithelial detachment. None of these parameters transmitted more than one-quarter of the actual maximum information given by gingival redness estimates. There is a strong indication that pocket depth has much less association with gingival redness than gingival stippling or gingival recession. The epithelial detachment level tended to transmit slightly less information with regard to gingival redness estimates than gingival recession alone when equal classifications were used. Due to the paucity of subjects demonstrating gingival bleeding, the AIT gives very little indication of its association with gingival redness. According to the original classifications, there was a strong relationship between pocket depth and gingival recession measurements as judged from the AIT value (Table 14). When only four categories were used, such association decreased markedly.

As the severity of the gingival redness increased, less stippling could be found. While the disappearance of gingival stippling is not necessarily associated with gingival redness, when the former is present on whole attached gingiva, the latter is seldom detectable. In subjects who demonstrated stippling on part of the attached gingiva, redness either did not appear or was restricted to only part of the attached gingiva (see Table 16).

In the absence of redness, the gingiva did not bleed upon mechanical stimulation. When the gingiva appeared red in colour, the majority of subjects still had no bleeding tendency. When subjects demonstrating considerable extension of gingival redness (score 1 to 3) were considered, the relationship between the degree of gingival redness and bleeding was again very weak (Table 17).

Table 18 shows that there were many subjects whose gingivae had receded 3.0 mm apical to the cemento-enamel junction, yet showed no gingival colour changes. The data also indicate that from one-half to nearly two-thirds of the subjects whose gingival margin was on the coronal portion of the tooth still manifested mild to moderate extension of the redness. However, there is a positive correlation between these two parameters. Such a relationship was more apparent if only subjects whose gingivae demonstrated redness were considered.

A positive assodiation is also apparent between gingival redness and pocket depth (Table 19). When pocket depth was 0.5 mm or less, the majority of the marginal gingivae and all the attached gingivae showed negligible redness. As pockets deepened from 1.0 to 4.5 mm, the gingival area demonstrating red colour change also increased. There were exceptions to this trend.

Table 16

The Relationship Between Gingival Stippling and Gingival Redness

Gingival redness	Gingival stippling -	+	++	Total
	Based on median estimates			
0	11	26	5	42
1	41	15	0	56
2	19	0	0	19
3	8	1	0	9
Total	79	42	5	126
	Based on observer C estimates			
0	10	20	14	44
1	22	19	1	42
2	15	1	0	16
3	24	0	0	24
Total	71	40	15	126

Table 17

The Relationship Between Gingival Bleeding and Gingival Redness

Gingival redness	Gingival bleeding 0	1	2	3	Total
	Based on median estimates				
0	42	0	0	0	42
1	51	4	1	0	56
2	16	2	0	1	19
3	7	1	1	0	9
Total	116	7	2	1	126
	Based on observer C estimates				
0	44	0	0	0	44
1	36	2	2	2	42
2	15	1	0	0	16
3	20	3	1	0	24
Total	115	6	3	2	126

Table 18

The Relationship Between Gingival Recession Level and Gingival Redness

Gingival redness	Gingival recession level (mm)				
	<0	0.0-1.0	1.5-3.0	3.5+	Total
	Based on median estimates				
0	14	22	6	0	42
1	26	26	2	2	56
2	5	4	8	2	19
3	0	1	5	3	9
Total	45	53	21	7	126
	Based on observer C estimates				
0	31	10	3	0	44
1	27	14	1	0	42
[illegible]	14	2	0	0	16
3	0	6	12	6	24
Total	72	32	16	6	126

Table 19

The Relationship Between Pocket Depth and Gingival Redness

Gingival	Pocket depth (mm)				
redness	0.0-0.5	1.0-1.5	2.0-2.5	3.0+	Total
	Based on median estimates				
0	10	28	4	0	42
1	6	34	14	2	56
2	1	7	10	1	19
3	0	4	3	2	9
Total	17	73	31	5	126
	Based on observer C estimates				
0	6	34	3	1	44
1	2	23	14	3	42
2	2	6	7	1	16
3	0	13	6	5	24
Total	10	76	30	10	126

Practically none of the pockets deeper than 3 mm were associated with normal gingival colour.

Table 20 shows a positive association between the degree of epithelial detachment and extension of redness on marginal and attached gingivae. In persons whose epithelial attachment was on or coronal to the cemento-enamel junction, about 50% demonstrated no gingival redness and most of the others demonstrated redness on only a part of the marginal gingiva. In none of the subjects whose epithelial attachment was less than 1.0 mm apically to the C-E junction, did the attached gingivae demonstrate a red colour change. Subjects whose epithelial attachment was located 2.5 mm or more apically to the CEJ, commonly showed more extensive colour changes involving the whole marginal gingiva and even some attached gingiva.

In subjects whose gingival or periodontal pockets were 0.5 mm or less in depth, the gingival margin did not recede beyond 1.0 mm apically to the CEJ. When the pocket depth was 1 mm to 2.5 mm, the majority of cases still showed 1mm or less of gingival recession. In the subjects with pockets 3 mm or deeper, chances of having greater gingival recession increased considerably (see Table 21).

In summary, the association between gingival redness and each of gingival stippling, gingival recession, pocket depth and epithelial detachment estimates was rather weak though the two-way distribution of each pair was far from random. Similar relationships also existed between gingival recession and pocket depth measurement. The association between gingival redness es-

Table 20

The Relationship Between Epithelial Attachment Level and Gingival Redness

Gingival redness	Epithelial attachment level (mm) ≤0	0.5-1.0	1.5-2.0	2.5+	Total
	Based on median estimates				
0	7	20	8	7	42
1	7	21	19	9	56
2	1	2	5	11	19
3	0	0	1	8	9
Total	15	43	33	35	126
	Based on observer C estimates				
0	26	8	5	5	44
1	14	10	13	5	42
2	7	2	5	2	16
3	0	0	3	21	24
Total	47	20	26	33	126

Table 21

The Relationship Between Pocket Depth and Gingival Recession Level

Gingival recession level (mm)	Pocket depth (mm) 0.0-0.5	1.0-1.5	2.0-2.5	3.0+	Total
	Based on median estimates				
<0	9	23	13	0	45
0.0-1.0	8	35	10	0	53
1.5-3.0	0	13	6	2	21
3.5+	0	2	2	3	7
Total	17	73	31	5	126
	Based on observer C estimates				
<0	8	45	15	4	72
0.0-1.0	2	18	10	2	32
1.5-3.0	0	11	3	2	16
3.5+	0	2	2	2	6
Total	10	76	30	10	126

timates and gingival bleeding tendency was negligible. According to the AIT calculated from the data of the original classifications for each parameter, the relationship with gingival redness estimates was greatest for epithelial detachment followed in decreasing order by gingival recession, gingival stippling, pocket depth and gingival bleeding estimates. If equal numbers of severity categories were used, gingival redness estimates were about equally correlated with parameter estimates of gingival recession, gingival stippling and epithelial detachment, and were associated less strongly with pocket depth measurements and again, least with gingival bleeding estimates. The relationship between gingival recession and pocket depth measurements was weak when measurements of both parameters were regrouped into four categories. Two major variations from the correlation trend are responsible for the weak relationship in each parameter pair. First, too much deviation at the two extremes of the severity scale, for example, the absence of gingival stippling and/or presence of marked gingival recession without simultaneous presence of gingival redness. Second, a fair degree of variation in the middle range of severity, for example, in subjects whose gingivae showed mild or moderate bleeding and/or 1.0 to 2.5 mm of gingival recession had a similar tendency to demonstrate varying degrees of gingival redness.

Chapter 5

DISCUSSIONS

5.1 On prevalence data

Since prevalence for individual periodontal disease parameters rather than index of gingivitis or periodontal disease was assessed in this study, it is almost impossible to compare the prevalence data of this study with those of others. The differences in the origin of study population as well as inherent inter-observer variation in estimating and measuring the periodontal disease prevalence and severity would further reduce the validity from such comparison. For example, in their study on the employees of Ontario Highway Department, Freedman et al reported the assessed Russell's PI scores on the upper first and second molars being, in average, 2.34 and 2.54 respectively (116). These data indicate only that the average severity of the periodontal disease in those employees is something more severe than gingivitis alone. In the study made by Mehta et al on Ontario civil servants, clinical diagnoses such as periodontitis simplex, necrotic periodontitis and periodontitis complex were used in the prevalence assessment of the periodontal disease (289). Hence, results from these two Canadian studies simply can not be used for comparison with those from the present study.

If gingival redness and/or swelling is taken as a measure of gingivitis, then approximately one-third of the study subjects were free from gingivitis. Based on median data, only one-eighth of the subjects showed no sign of epithelial detachment. These data suggest that, in many cases, while the gingivae showed no observable colour or texture changes, the epithelial attachment had already migrated apically. This finding is observable mainly in some dental student participants whose gingivae though have receded apically to the C-E junction are in perfectly normal colour and texture. Data from sampled U.S. adult population reveal that while one-quarter of subjects are free from any periodontal disease, only another quarter of subjects have true periodontal pockets, i.e., epithelial detachment (202,212). Hence, it seems to suggest that present study subjects have greater prevalence in epithelial detachment than the representative U.S. adult population. Such comparison is however subjected to serious error because the origin of study populations and the age distributions between two groups differ greatly. The small number in each age group of present study subjects does not allow for comparison of the age specific prevalences between two study populations. The judgement of the epithelial detachment may also differ between two studies that make any comparison completely meaningless. To generalize the prevalence data from this study subjects who sought for emergency dental treatment, to the population which they belong to may also cause serious misleading. Since the purpose of present study is to estimate the inter- and intra-observer variation in each parameter estimate or measurement, slight variations from the general population should not seriously affect its validity for generlization.

According to the original plan, standardization (calibration) among three observers in measuring each parameter was to be made prior to the actual data collection. Such standardization has been shown to improve inter- as well as intra-observer agreement in periodontal disease measurement (77,356,416). The course of such standardization was however cut short due to very few subjects willing to be studied. Therefore, the author resorted to frequent explanation to other two observers regarding each parameter score. Such alternative effort proved to be not totally satisfactory as reflected in prevalence data assessed by the three observers. For example, lack of unanimity in identifying the anatomical demarcation between the marginal and attached gingivae causes observer C to record more attached gingivae being in red colour than those found by observer A and B. Inconsistence in locating the C-E junction also results in great variation in recording the prevalence of gingival recession and epithelial attachment level, namely, observers A and B over-estimated them while observer C underestimated. Thus, of 126 subjects, epithelial detachment was respectively recorded by observer A and B in 118 and 111 subjects, its was 80 subjects according to observer C's assessment. With such big differences in three observers' assessments, the median prevalence of this parameter (109 in 126 subjects) provided limited usefulness for comparison with findings from other studies.

The results indicate that nearly two-thirds of all subjects had gingival redness and yet only a few of them had gingival bleeding. Such phenomenon cast some doubt on the diagnostic procedures which emphasize gingival bleeding as the main criteria in gingivitis index (249).

The negligible frequency with which gingival ulceration and gingival or periodontal pocket pus was observable under the described condition in this study, despite the reasonable distribution in other parameter scales, suggests that either the detection methods were not sensitive, the parameter can occur only in much more severe stage of the disease than that was observed in this study population, or these two parameters are of little usefulness for the development of the periodontal disease index. occasionally,it was difficult for some observers to differentiate the pocket pus from tissue exudate which frequently mixed with plaque substances while emerging from the pocket. Such a diagnostic problem contributes to the variation among three observers in pocket pus prevalence estimation.

5.2 On inter- and intra-observer variation in each periodontal disease parameter estimate or measurement

5.2.1 On gingival stippling estimate

There has been no study which assesses the inter- or intra-observer variation in gingival stippling estimate. Results from this study indicated that successful replication among three observers and within each observer from re-examination was made in 75% or more of the cases. Such a relatively good result seems to be due to the fact that more than half of the study subjects had no grossly detectable gingival stippling— an estimate on which all the observers or the same observer is likely to agree. In their study on the pattern and distribution of gingival stippling, Rosenberg and Massler reported that the depressions which gave the stippled appearance were barely visible to the naked eye, but are readily apparent under three to five times magnification (348). Such diagnostic difficulty has not been reported by others nor was particularly experienced by the present examiners. It is therefore unlikely that the inter- and intra-observer variations in stippling estimate of this study are the consequency of a deficient diagnostic procedure.

Despite the fact that frequency distribution of deviation data of each observer estimate from the reference estimate and of the second from the first estimate by the same observer shows a relatively good inter- and intra-observer agreement, the average amount of information transmitted (AIT) in each occasion was less than one-half of the actual maximum information available. This can be partly explained by the skewed frequency distribution of this parameter and the degree of deviation in relation to only three severity classifications for this parameter.

The major difference between the frequency distribution of deviation data and the statistics of the average amount of information is that the former considers only the occurrence of net deviation, whereas the latter takes into account both net deviation and the estimates such deviation actually taking place. For example, the deviations of score 1.5 from 3.0 and of 0 from 1.5 make no difference for the frequency distribution of deviation, but provide different information when AIT is considered, because they give different two-way distributions from which the statistic of the average amount of information is estimated. This explains the apparent contradiction that observer A who, though seemed to achieve, the best scoring reproducibility if judged from frequency distribution of the deviation data, actually transmitted the least amount of information in comparison with observer B and C. This phenomenon appeared not only in gingival stippling estimate but in other parameter estimates and measurements as well.

5.2.2 On gingival redness estimate

Although no one has ever tried to assess inter- or intra-observer variation in the estimate of gingival redness itself, observer variation in gingivitis assessment has been evaluated by some investigators. Since practically in all these studies, the gingivitis was diagnosed according to the presence or absence of the gingival redness and/or swelling, it seems to be most appropriate to compare the observer variations in gingival redness estimate of this study with those in gingivitis estimates by others.

In an epidemiological training course, Davies et al observed that the examination on the periodontal disease, especially the gingivitis was most unreliable. In 416 teeth of 13 patients who were examined by each of 13 examiners, the number of teeth being diagnosed to be associated with gingivitis ranged from 22 to 246 according to the observers (82). The present study seems to show much better inter-observer agreement based on the prevalence data which are 81,83 and 82 in 126 subjects respectively for observers A,B, and C.

Sandle evaluated the inter-observer variation in P-M-A scoring in which each of the eight participant dentists examined the same 10 patients. The results showed that in about 50% of P,M and A assessment did the differences among the examiners' estimates occur more than by chance (370). Recently, using DHC's Gingivitis Index (Dental Health Center) for studying observer variation, Suomi found that the differences in mean gingivitis score between any two of three observers ranged from 0.15 to 0.17 (432). These data, however, provided little information about the actual inter-observer variation because cases which were over- and under-estimated will be cancelled out in the course of averaging the difference. Furthermore, from the results of present study, there is much doubt about the justification for making an average of the variations or deviations because their distributions hardly ever approach to the Gaussian. Hence, it is very difficult to compare the results from Suomi with those from the present study.

The major source of inter-observer variation in the present study lies in deciding the location of gingival redness either within the marginal gingiva or extending to the attached gingiva. For this reason, observer C in comparison to observer A and B reported more subjects with score of 3. Variation arised from this reason could have been greatly reduced if better standardization and calibration had been made prior to data collection.

From the same epidemiological training courses, Davies <u>et al</u> (82) also found very poor replication in gingivitis estimate made by each of the 13 participant examiners. In a total of 416 teeth, one examiner recorded the presence of gingivitis in 22 and 80 teeth respectively on the first and second day of examination.

Even the examiner who achieved the best intra-observer agreement in gingivitis estimate made the identical score in only 56.3% of the cases. The consistency ratio for gingivitis estimate (which is defined as the percentage of the number of occasions in which gingivitis was recorded as present at both occasion in the total number of occasions in which gingivitis was recorded as present at least once) ranged from 18.7 to 76.4%. The corresponding ratios for the present three observers ranged from 83.3 to 93.2%. Comparing these percentage with the findings from the frequency distribution of deviation data and the statistic of the average amount of information, it is clear that the "consistency ratio" is a much less sensitive measure for intra-observer variation.

Smith <u>et al</u> demonstrated that an identical gingivitis estimate could be made on 84% of replicates using Ramfjord's Gingivitis Index and in 82.4% of replicates using DHC Gingivitis Index (416). Such a very good intra-observer agreement is possibly attributed to the relatively healthy gingivae in their study subjects (50 male recruits at U.S. Naval Training Station) and to the fact that the subjects were re-examined only one hour following the initial examination. For such a short lag between two examinations, the gingival tissue does not likely alter and examiner may still have quite fresh memory from the initial assessment. Suomi <u>et al</u> also examined institutionalized young adults. On each morning four subjects chosen randomly from all participant subjects were re-examined one hour following the initial assessment. The identical gingivitis estimate was made by observer A in 81% of the teeth which improved to 98% on the fifth morning. The corresponding data for examiner B were 89 and 93% respectively on the first and fifth morning (435). Considering the simplicity of the DHC Gingivitis Index they used, the interval between two examinations and the number of subjects (and teeth) re-examined each morning, such a good intra-observer reproducibility may not be too difficult to achieve. The identical gingivitis estimates were made in this study in 77.3,81.8 and 70.5% of the cases by observer A, B and C respectively. Such results may not be too poor in comparison with the findings hitherto presented because the longer interval between repeated assessments as used in this study not only reduce the possibility of observers' memory but also involved the fluctuation of the gingival tissue condition. Hoover and Lefkowitz reported the fluctuation of gingival areas in 25 dental students (i.e., the percentage of the areas changed from normal to disease or disease to normal) in each two-week period remained relatively constant at approximately 24%. Only in 40.9 of 501 initially inflammed areas and 40.9% of 903 initially normal areas remained the same condition throughout the ten-week experimental period (174). Approximately 20% of such fluctuation in each four-week period were observed by Suomi <u>et al</u> (435). To an unknown extent, those reported fluctuation might have actually been resulted from the intra-observer variation alone, rather than from real fluctuation. It is also very clear that all those fluctuation data were based on the presence or absence of gingivitis only. It is not certain what extent of fluctuation of gingivitis would be if the changes in severity had also been considered. The comparatively older ages in present study subjects than those of other studies may also increase the inter- and intra-observer variation because more subjects had gingivitis,for which severity the observers are poorer to agree with than for a healthy gingiva.

There is systematic variation in this parameter estimate between observer A and B on the one hand and observer C on the other. In contrast to observers A and B, observer C tended to systematically overestimate the severity of the parameter. For this reason, despite the poor inter-observer agreement, each observer achieved relatively the same degree of intra-observer agreement.

The findings and discussion hitherto presented also cast serious doubts about the validity of the conclusions drawn from some clinical trials on the relationship of various dietary supplements and the gingival health (50,60,61,229,233,339,346). The comparatively short period of experiments, mostly three days only, and few study subjects may have precluded any possible blind assessment because of memory effect. The slight difference between the experimental and placebo (or simply control) groups in mean gingivitis scores at the end of the studies may well be the results of intra-observer variation in gingivitis estimates unless such variation affects both groups equally.

To extend the experimental period, on the other hand, would likely to involve the natural fluctuation of the gingival tissue condition. It will also increase the intra-observer variation in gingivitis estimate as a result of lacking in scoring practice. However, most of these variations can possibly be resolved if initial matching in pairs between the experimental and control groups is carried out according to the gingivitis severity, and if several observers examine each subject throughout the study. The former provision would prevent or reduce the degree of unequal gingival fluctuation taking place in the course of experiment in two groups. Taking an average or median value of several observers' estimates for a subject would to some extent reduce the unusual great intra-observer variation from repeat assessments by a single observer. Such variations are unlikely to be in the same direction among observers, thus, cancel out altogether.

5.2.3 On gingival bleeding estimate and judging the presence or absence of the gingival or periodontal pocket pus

Since there were only a few cases (less than 10%) in which the gingivae showed bleeding following varying intensities of stimulation and even fewer cases (less than 5%) in which the gingival or periodontal pocket pus was clinically detactable, the likelihood of inter- and intra-observer agreement became very great. Thus, for gingival bleeding estimate, observers A, B and C achieved an identical estimate with the reference estimate in 98.4, 97.6 and 90.5% of cases. However, if only cases which were once, at least, scored 1 or greater according to the reference or observer estimates, were considered, the identical estimates were achieved by observers A,B and C in 83,2, 75.0 and 25.0% respectively. The corresponding three observers' data from intra-observer comparison are 86.4, 86.4 and 95.5% for all cases, and 0, 14.3 and 33.3% for cases which were once, at least, scored as positive. Therefore, it seems apparent that the consistency ratio was rather poor and that the major contribution to inter- and intra-observer agreement comes from the cases in which the gingivae did not bleed at all. These same phenomena of observer variation also occurred in pocket pus assessment.

Considering the fact that there are only a few cases in which the gingival bleeding and pocket pus were clinically detectable and rather poor inter- and intra-observer agreement among these few cases with positive signs, to conduct a clinical trial aiming to improve the gingival health by assessing these two parameters alone is not likely to be a rewarding study (427). To include and emphasize these two in addition to other parameter estimates will not likely supply one with additional informations either.

The statistic of the average amount of information became less useful for the parameter which has very low prevalence. Based on the mathmatical calculation of this statistic (see appendix II), a frequency distribution of such low prevalence results in two major disadvantages; first, the actual maximum information available is less than with a fairly equal distribution for all severity scores; second, the information lost would be little only as long as, in the case of inter-observer variation, each observer estimate does not result in any further increases to the group having the greatest frequency according to the reference estimates, (or, in the case of intra-observer variation, the second assessment does not result in any further increase to the group having the greatest frequency according to the first assessment), otherwise, the information lost would be tremendous. For example, during the initial gingival bleeding estimates in the 44 subjects, the reference estimate was 0 in 40 subjects. There were two cases in which the gingival bleeding was overestimated by observer B and was underestimated by observer C. Therefore, the number with a 0 score was 38 and 42 according to observer B and C estimates respectively. However, the information lost was 0.09 and 0.26 binary digit respectively for observer B and C.

The application of the statistic of the average amount of information transmitted as an inverse measure for inter- and intra-observer variation of pocket pus estimate was further limited to the point of completely useless. Two factors are responsible for such limitation; first, the prevalence of pocket pus was even lower than that of gingival bleeding; second, while there are four severity classifications for gingival bleeding, only two categories are assigned to the pocket pus.

5.2.4 On measurements of the gingival recession, pocket depth and epithelial detachment

From an epidemiological training course, Davies et al (82) reported that the prevalence of periodontal pocket as assessed by 13 examiners on the same 13 subjects was totally unreliable. The total number of pockets recorded by the observers ranged from 4 to 29 during the first assessment and from 2 to 19 during the second assessment. Their results also indicated that the variability between examiners in assessing pocket formation was as much as the actual variation of pocket formation in the patients. In the same study, each examiner re-examined the same 13 subjects on the following day, and their consistency ratio ranged from 0 to 26.3% indicating very poor intra-observer agreement. Such low consistency ratios are possibly the result of relatively low prevalence of true periodontal pocket in their study subjects, that a slight difference in measuring pocket formation on potentially positive cases would greatly reduce the consistency ratio. Result from present study in epithelial detachment measurement which is equivalent to the pocket formation in the study by Davies et al, showed much better consistency ratio by three observers (93.2, 90.7 and 83.3).

Suomi (432) reported that the mean pocket depth (in fact, epithelial attachment level) as measured by any two of three observers on the same ten subjects differed for 0.17 to 0.60 mm. These mean differences are rather great if one considers that the mean pocket depth of the study population, from which 10 subjects were randomly chosen for study of inter-observer variation, was 0.83 mm only, and that mean difference rather than standard deviation of individual differences (method error) was estimated. Repeat assessment for 20 subjects on successive morning by the same observers resulted in difference in mean pocket depth for 0.12 to 0.21 mm depending on observers. Studies made by Davies et al (82), Suomi (432) as well as by Castenfelt (48) all revealed a systematic variation among observers in measuring the pocket depth and epithelial attachment level. Such variations persisted even following intensive effort of standardization and calibration among the examiners. This phenomenon was apparent also in the present study in that observers A and B in comparison to observer C systematically over-measured both gingival recession and epithelial attachment level.

Recent study made by Smith et al on 50 male recruits at U.S. Navy Training Station showed that very good intra-observer agreement on the measurements of the gingival recession, pocket depth and epithelial attachment level could be achieved. The percentages of the agreement for the three parameters respectively were 82.3, 81.2 and 94.5 (416). Such a good reproducibility is possibly attributed to relatively short interval between two assessments (one day) and rather healthy periodontal tissue in their study subjects (mean values for three parameters were -2.25, +2.31 and +0.06 mm). Since Smith et al recorded the measurements in complete millimeter whereas one-half millimeter intervals were recorded in this study, to make our data comparable to theirs, the percentages of assessments with repeat measurements differing within ±0.5 mm were pooled together. Thus, the percentages of agreement in replicate measurements by observer C were 77.2, 77.2 and 79.5% respectively for the same three parameters. The corresponding data for observer A were 72.8, 84.1 and 54.4% and for observer B were 95.5, 86.4 and 77.2%. Hence, intra-observer variations in the three parameter measurements were greater in this study than those by Smith et al. One possi- explanation is that in comparison to the study by Smith et al, present study subjects had relatively more severe periodontal disease.

Glavind and Loe (135) once reported that the method errors by the same observer in measuring the pocket depth on the buccal surface of the upper right and left first molars were 0.26 and 0.17 mm respectively. The corresponding data for epithelial attachment level were 0.26 and 0.39 mm. In comparison to the same data in this study, it is apparent that Glavind and Loe are better than us in intra-observer agreement for these parameter measurements. Yet to what extent the rather short interval between replicate assessments (15min.) they selected,and the relatively healthy periodontal tissue of their study subjects (20-30 years of age) contribute to such difference is not certain.

Results from the present study as well as from others hitherto presented with respect to the intra-observer variation, expressed as method error, for the parameters of pocket depth and epithelial detachment suggest that a reduction in sulcus depth from an average of 2.3 to 2.1 mm or from 2.2 to 2.0 mm following four-day protein supplements (51,341) or from an average of 2.20 to 2.13 mm or 2.12 to 1.96 mm following four-day regular or sustained-release multivitamin supplements (54) may not mean anything other than intra-observer variation in repeat assessment of the parameter. In comparison to the gingival recession and pocket or sulcus depth, epithelial attachment level is a more stable parameter for the measurement because it is measured from a fixed anatomical landmark, i.e., C-E junction. Its apical migration along the root surface is however in most cases very slow. Hence, for a longitudinal study such as a clinical trial,it should be carried out for a fairly long period of time to allow for enough parameter changes to take place, so much so that the disturbing effect of intra-observer variation is greatly reduced. To study the control pairs simultaneously would also eliminate the systematic variation in subsequents assessments by the same examiner.

As stated in the discussion section on prevalence, observers A and B tended to record the gingival margin as being at the C-E junction (recession 0 mm), while in some cases observer C found one-half to one millimeter of the crown coronal to C-E junction covered by the gingival margin (recession -0.5 to -1.0 mm). This inter-observer difference in gingival recession also reflected in epithelial attachment level measurement, namely, observer C markedly undermeasured it while observer B and particularly observer A overmeasured it. Hence, when each observer measurement was compared with the reference measurement which was the median of three observer measurements, the deviation was much small for observers A and B and greater for observer C. This again reflects in the same manner for the observers' variation on the estimate of method error.

For any specific measurement or estimation, each examiner, in general can agree better with himself than with others. This phenomenon has been demonstrated in the assessment of gingivitis and pocket depth by Davies et al (82) and Suomi (432). However, results from the present study showed greater intra- than inter-observer variation in most parameter assessments. This is possibly resulted from the fact that in contrast to only one day interval between replicate assessments in those two studies mentioned, one to two weeks interval in general, was employed in present study. Thus, marginal gingival tissue alteration caused by inflammatory changes during the course of the study may actually have,to a certain extent, affected the gingival recession and gingival or periodontal pocket depth. The epithelial attachment level, however, was much less affected by the study period because the epithelial attachment or pocket bottom is unlikely to change within such a period of time. The fact that each observer's method error in epithelial detachment measurement is very much smaller than the the sum of the method errors of his own gingival recession and pocket depth measurements (observer C, for example, they are 0.55 mm against 0.47 and 0.46 mm) also substantiated this explanation.

Since many more severity classifications were assigned for the measurement of gingival recession, pocket depth and epithelial detachment than for other parameters previously discussed, both the actual maximum information available and the information transmitted greatly increased. This finding supports the conclusion made by Liddell in his study on inter- and intra-observer error in x-ray readings (237).

5.3 On the association in each selected pair of the parameters

A weak association between gingival redness and each of gingival stippling, gingival recession, pocket depth and epithelial attachment level found in this study is not unexpected. Absence of stippled appearance in the presence of normal gingival colour has been reported (152). Same study also revealed that gingival stippling did not reappear following a successful gingivectomy treatment. In some of present study cases, gingivae receded apically to C-E junction without concomitant colour changes suggesting causes other than inflammatory origin, such as faulty toothbrushing method, or that the inflammation had subsided following either better oral hygiene regimen and/or professional treatment. If cases which had these particular case histories had been identified and excluded from the estimation for the parameter pair relationship, the natural correlation between gingival redness and gingival recession is likely to be much greater than that previously presented. In some cases with very chronic periodontal disease, the connective tissue under epithelium becomes so fibrotic that the gingival colour can not be easily differentiated from the normal pink colour. Thus, some cases with fairly deep pockets showed no gingival redness, while other cases with very shallow gingival sulcus presented very extensive colour change. These cases contribute to the rather weak relationship between gingival redness and pocket depth. Since epithelial attachment level is the sum of the estimates of gingival recession and pocket depth, its association with gingival redness is, therefore, the function of the association between its component parameters and gingival redness.

As has been demonstrated in the section of Results, the strength of the association between parameters strongly depends on the numbers of the severity categories in each parameter. With a constant relationship between two parameters, the more categories a parameter has, the greater average amount of information that parameter estimates can transmit from the another parameter estimates. In the course of the study the author have observed the possibility to estimate gingival redness according to its intensity in addition to anatomical extension. To do so would apparently involve some subjective judgement, yet likely to improve the correlation estimates between this parameter and others. Such scoring method would also reduce the inter- and intra-observer variation because the average amount of information transmitted, which is an inverse measure of observer error (variation), increases as number of catergories for the parameter increases (237). It would be a worthwhile study to determine the practicability of such scoring system for gingival redness and to reassess this parameter's relationship with other parameters as well as the observer variation in this parameter assessment.

The relationship between gingival recession and pocket depth is stronger than other parameter pairs when both parameters are measured at 0.5 mm interval. Such relationship would be even stronger than which was presented if cases whose gingivae receded apparently as a result of faulty toothbrushing techniques and cases who had received, prior to examination, a successful treatment for chronic gingivitis or periodontitis had been excluded from the relationship assessment. In the former cases, the gingivae receded with little concomitant changes in pocket depth. In the latter cases, the treatment resulted in slight gingival recession and reduction in pocket depth due to disappearance of the pseudo- or true pockets.

There is a rather weak relationship between gingival redness and bleeding. It suggests that with severity based on anatomical extension alone, the gingival redness gives little indication of bleeding tendency. Intensity of the gingival colour change may be more associated with the bleeding tendency. Improving sensitivity for gingival bleeding detection, inserting a filter paper into the gingival sulcus or periodontal pocket for example, may increase the prevalence of this parameter which in turn will improve its correlation estimation with gingival redness if their relationship indeed exists.

Since there are some relationships among these periodontal disease parameters any clinical trial or epidemiological study which intends to investigate these parameter conditions has to deal with them simultaneously. Any inferential statement concerning one parameter will consequently affect the inferences to be made for other parameters. Hence, to make separate report for each periodontal disease parameter from a study dealing with several parameters is totally unjustified (49-51,53-57,59-62,99-101,339,340,342). For the same reason, to construct a periodontal disease index with these component parameters, one has to take inter-parameter relationship into account by means of multiple regression technique or the analysis of principal component.

Chapter 6

CONCLUSIONS

From both the review of existing knowledge and the first phase of index development, the following conclusions seem to be approprate.

i. A new periodontal index which is specifically suitable for longitudinal studies of periodontal disease is needed for clarifying some currently contradictory findings with respect to the possible roles of nutritional and systemic factors in the initiation and progression of the periodontal disease, and for direct assessment on the cause and effect relationship.

ii. Further refinement in estimating gingival redness and, especially, gingival bleeding and pocket pus seems to be necessary for greater sensitivity and less observer variation.

iii. Since both inter- and intra-observer variations are very small in estimation of gingival stippling and gingival redness and in measurement of gingival recession level and pocket depth, these four parameters are suitable for further index development.

iv. The apparent interrelationship existed among these four periodontal parameters has to be taken into account in construction of the index.

v. Based on observer variation in gingival stippling estimate and epithelial attachment level measurement, the validity of many conclusions made from some clinical trials with respect to the beneficial effects of various nutrient supplements on these parameters health is very questionable.

APPENDIX I

CRITICAL REVIEW of EXISTING KNOWLEDGE

(The references quoted in this review are presented in the section of BIBLIOGRAPHY).

I.1 Introduction

Epidemiology is the study of the distribution and determinants of disease frequency in man. Two main areas of investigation are indicated in this definition - the study of the distribution of disease and the search for determinants of the observed distribution. The first area, describing the distribution in terms of age, sex, race, geography, ets., might be considered an extension of the discipline of demography to health and disease. The second area involves explanation of patterns of distribution of a disease in terms of causal factors (262). Under the heading of epidemiology may also be placed the clinical trial, the purpose of which is to ascertain to what extent a preventive measure, a therapeutic method, or a particular drug may change the course of a disease for the better or worse (462). This review will focus mainly on epidemiologic knowledge of periodontal disease.

Most current information about the distribution and severity of periodontal disease in population groups has been collected within the past fifteen years. Mehta <u>et al</u> (288) in their review found little to present beyond clinical impression. Since then, a series of field indices has been developed which will be discussed in detail at the latter part of this review. All are devices for quantitating data so that statistical analysis may be employed. To date at least some indices have been useful for comparisons of population prevalence and severity, while others have been suitable for clinical experiments. Following the development of the Oral Hygiene Index (156) and the Simplified Oral Hygiene Index (157), which are used to estimate amount of oral debris and calculus, dental deposits on teeth and age have been reported to account for the major proportion of variation in severity of periodontal disease among populations of distinct demographic character. The epidemiological findings cast doubt on traditionally held significance of nutrition, systemic health and disease, malocclusion and trauma from occlusion in the initiation and progression of periodontal disease. These data also contradict the results from clinical trials relating various nutritional and dietary factors to periodontal disease.

The following review will attempt to fulfil four objectives: 1) to provide current accepted knowledge concerning the epidemiological pattern of the periodontal disease, 2) to present the contradictory or confusing conclusions which exist between clinical impressions, clinical trials and population studies concerning the relationship between nutrition, systemic health, and periodontal disease, and, if possible, to explain the reasons for the discrepancy in terms of study designs, data treatment, interpretation of data and so on, 3) to point out possible deficiencies in the taking of oral deposits as evidence of neglect of oral cleansing as well as the fallacies inherent in standardizing the factors of oral deposits ("oral hygiene") and age, whenever correlation of periodontal condition with other factors is estimated, 4) to explain why longitudinal population studies are essential to better investigate periodontal disease, clarify the current controversial conclusions, and to provide reasons why there is a need to design a new index suitable for these studies.

I.2 Morbidity and severity data for periodontal disease

In assessing periodontal disease two morbidity statistics have been used - incidence rate and point prevalence rate. The incidence rate is defined as the number or new illnesses beginning within a specified period of time, related to the average number of persons to risk during that period, whereas the point prevalence rate is defined as the number of illnesses existing at a specified point of time and related to the number of persons exposed to risk at that point of time (168).

There is very little data about the incidence rate of periodontal disease. Parfitt's five year longitudinal study of gingivitis in England (316) and Hoover's study of the fluctuation in marginal gingivitis (174) are exceptional examples. If deviation from perfect dental health is used as a yardstick, practically all human beings have periodontal disease (462). But one person may have a slight gingival inflammation in a single gingival unit as compared with another one in whom most of the supporting structure around all the present teeth is broken down. The public health significance between these two cases differs greatly. It is often useless to compare the prevalence

of periodontal disease in different populations as prevalence of some kind will be close to 100 per cent in most populations. To increase the comparability, severity, as well as indices to measure it, had been introduced and become the major measurement of periodontal disease. Severity of periodontal disease is sometimes expressed in prevalence rates of different stages of periodontal condition. For example - among the 90 million adults with teeth, about one in four had destructive periodontal disease (with pockets), two in four had gingivitis ranging from mild inflammation involving a few teeth to severe inflammation involving all teeth (with periodontal disease, but no pocket) and the remaining one in four exhibited no signs of periodontal disease (212). Severity may also be measured as average number of pockets per person and per tooth (289).
The terminology of prevalence, severity and incidence has been greatly abused and misused in the literature, and when incidence rate is mentioned in the articles, more often than not it is actually prevalence rate (9,23).

I.3 Relations between severity of periodontal disease and suspected etiological factors

I.3.1 Age

Prevalence and severity of periodontal disease, in general, increase with age. Since different parameters of periodontal disease have been employed in epidemiological studies, their association with age also differ to some extent.

As proportion of subjects with gingivitis was considered, most studies showed that it occurred at a very early age and reached a first peak at early teen and leveled off or declined thereafter until the late teens (20,278,316). Studies which included adult populations also demonstrated a slow progression of gingivitis prevalence with increasing age (9,278). Tokelau islanders, however, did not show biphasic prevalence of gingivitis along the age scale and reached the maximum prevalence at the age group of 25-29 years (22). Quite contrary to these findings, Brown et al (40) found no consistent age trend of the same parameter when they studied school children of three Ontario towns. Similar findings among Bostonean adolescents and adults were mainly due to high proportion of subjects having gingivitis since early adolescence (273).

Severity of gingivitis usually progressed rapidly during childhood and reached the first peak between the age of 9 and 13 years (316,350,352). No such age trend was found by Brown et al (40). Benveniste et al also reported higher gingivitis scores among persons of 17 years or older than among those who were less than 17 years of age (30).

With one exception which observed the population of very narrow age range (288), the proportion of subjects with periodontal disease continued to rise with increasing age (17,26,37,83,202,212, 213,289,358,373,385,420). One study demonstrated it as a cumulative curve pattern with two inflections occurring at age 25 and 55 years respectively (373). Oral deposits, expressed as OHI scores or CI scores, were also found to increase as age increased (213,289).

Russell's Periodontal Index (PI) has been widely used for field studies since it was developed. With the few exceptions (105, 153,156,402), in which only populations of narrow age ranges were studied, the average PI scores increased consistently with age (1,17,18,22,25,73,93,103,109,116,153,154,159,164,202,212, 213,243,247,259,267,350,352,358,364,367,368,395,396,400,401,414, 463,469). The index did not increase at the same rate from one age group to another, and indeed, a sharp break could be observed between the age group of 15-19 years and 20-24 years among rural Maori population (22), between the age group of 15-19 years and 20-29 years among Ecuador civilians and Montana Indians (154), and between 15-19 years old and 20-29 years old among Ugandans (414). When oral deposits, measured as OHI and/or CI scores were assessed, corresponding increases in scores with age were found (109,154,159,164,213,243,259,368,395,400, 401,414,469). But such increases in OHI and/or CI scores could only partly explain the increase of PI scores with age due to the fact that even when PI scores of the subjects who had like OHI scores were considered, the older subjects, when compared with younger ones, still had higher average PI scores (213,247, 463). In one study, the age factor alone was reported to explain 47.2% of the total variance of the PI scores (368). With the exception of the Ontario school children study (283), prevalence of subjects having one or more periodontal pocket increased with age (17,20,202,212,213,247,330,358,400,402,410). Periodontal pockets may start to appear in people as early as 10 years of age (20,283,358), between age of 10 to 14 years (247,330,400,401) or between the ages of 15 to 19 years (17,400).

Since there might be observers' bias, such differences among various study populations may not be meaningful. There was also an indication of increasing rate of pocket prevalence during early adulthood (401) and during middle age (247,358). Corresponding rise of OHI and/or CI scores with age was also observed (213,330,400,401) with one exception being the study by Sheiham of Surrey school children (402). Like the prevalence of subjects with periodontal pockets, the average pocket depth per subject also became greater as age increased (30). Alveolar bone loss, which is measured from radiographs, was found to progress as people became older (16,375,394,413). The correlation coefficients between age and alveolar bone loss ranged from +0.37 (394) to +0.81 (413) depending on age ranges, size of samples, and possibly also other characters of the study populations.

There are some other parameters which also measure prevalence and severity of periodontal disease and were found to rise with increasing age in various populations. They were gingival recession rate (GRR) (18), percent of subjects having gingival recession (149), percent of subjects having both gingivitis and periodontal pockets (22), percent of subjects having chronic destructive periodontal disease (273), average numbers of pockets per subject (283,289), average numbers of pockets per tooth (289), and Slome's periodontal score among Jewish women (415), Ramfjord's Periodontal Disease Index scores (PDI) was found to progress with increasing age among adult population (439), but to have no consistent age trend among children and adolescents

(330.439).

There was some fluctuation in the proportion of children with abnormal gingivae between the ages of 5 and 13 years among school children of four Canadian provinces for which data are available (235,236). To cover as many aspects of periodontal disease as possible, it is worth mentioning that in South India, about one-half of the males and two-thirds of the females who had acute necrotizing gingivitis (ANG) were less than 10 years of age (322), and that in Bombay City, periodontosis mainly occurred among subjects of the age group 20-25 years (332). Although periodontal disease progresses in prevalence and severity with age, we still do not understand the responsible mechanism. Since surveys on adult populations in the U.S.A. revealed that about one-seventh of the males and one-fifth of the females at the age group of 55 to 64 years had no detectable periodontal disease, and that, for the same age group, only 45,6% and 35.5% respectively among the males and the females had true periodontal pockets (202,212), it seems unlikely that the aging process itself would initiate or promote periodontal disease. Instead, age may only an indirect indicator of the duration of the etiological factor or factors which may or may not relate to age, and of the duration of the periodontal disease since its inception.

I.3.2. Sex

Of the studies which consisted of both children, adolescents and adult populations, the periodental disease occurred, in general, more frequently and more **severely** among the males than females (1,22,116,149,159,164,202,212,258,273,289,368,420,430). At the same time, male subjects were found to brush their teeth less frequently than females (273), to have more persons, proportionally, with more oral deposits (289) and to score higher with regard to OHI, CI and/or DI (1,159,164,213,368). Studies among adult populations in the U.S.A. demonstrated, however, that differences in PI between sexes disappeared when comparison was made between sexes with like age and simplified OHI scores (213). Studies among rural Maori population did not reveal obvious difference in CI or DI scores between sexes, in spite of the fact that males had higher average PI scores than females for each age group (22).

Several studies has indicated no consistent sex difference at young ages, only slightly higher prevalence of periodontal disease among teenage girls than boys, and generally more significant differences in periodontal conditions among adult men and women (37,154,122,352,358,401,414). Oral deposits followed this same pattern in a Ugandan study (414), but were found to be consistently worse among males than females throughout all age ranges in two other studies (154,401).

Contrary to the above findings, studies in Ethiopia (247), rural central India (267), Sudan (103), among Bedouins in Israel (350) and Tokelau Islanders (22) demonstrated worse periodontal condition among boys than girls and among adult women than men. Males among Bedouins in Israel scored, for each age group, higher CI and DI than females (350). Waerhaug reported consistently higher average PI scores among Ceylonese females than males who had the same age and simplified OHI scores (463). Prevalence of periodontosis was shown to be higher among women than men in Bombay City (332). We do not understand exactly why female populations in general and adult women in particular had higher prevalence and severity of periodontal disease than the males of these populations (22,103,247,267,332,350,463), but it seems interesting to find that these study populations come from less developed geographic regions, and it was suggested by Waerhaug in his study among Ceylonese (463) that frequent child-bearings among these women, in contrast to women in more developed areas, might have adverse effects on periodontal health. But such a hypothesis remains to be proved.

Studies in Chileans (17,18), Melbourne suburbanites in Australia (243) and Bostoneans (273) revealed no consistent sex difference in the prevalence of periodontal disease. Arno and his associates observed no difference in the prevalence of gingivitis among male and female employees who belonged to the same age and oral deposit categories and smoked the same number of cigarettes (9).

When children and teenagers were considered, French Polynesians (20) and Ontario school children (40) were observed to have higher prevalence of gingivitis among boys than girls. No such obvious sex difference was found among Israeli rural children (352), Bedouin children in Israel (350), or Ibadan children in West Nigeria (105). Contrary to this, girls, as compared with boys, at Surrey school in Kingston had a higher average PI score, a higher proportion with pockets as well as a higher average OHI score (402).

Thus, there seems to be no definite sex difference in the prevalence and severity of periodontal disease, and when they did exist, most of such differences could be explained by intermediate factors, such as degree of oral deposits and cleansing. Although there are still some studies in which one can not explain the differences between the sexes in periodontal condition, one must also realize that the sample bias of these studies may have been so great that this itself may have caused such differences in periodontal disease prevalence.

I.3.3 Geographic distribution and ethnic factor

Epidemiological studies of the prevalence of periodontal disease have been made on populations from more than 50 geographic areas (1,17,20,22,38,73,93,103-105,109,110,116,153,154,159,163, 164,180-196,201,202,212,230,235,236,243,247,259,267,273,279, 282,283,288,289,322,330,350,352,358,361,363,364,367,368,400, 401,402,414,415,461,463,469). To obtain a rough idea about the prevalence and severity of the disease among populations of various geographic regions, these studies are summarized according to:

(A) Prevalence of periodontal disease
(B) Prevalence of gingival abnormalities
(C) Percentage of subjects with one or more pockets, and
(D) Mean PI score (Russell's PI index)

To compare the prevalence of periodontal disease among different populations assessed by different observers was always handicapped by great observer variation, even when the same index was employed (82). And there is no way to make a

(A) Prevalence of Periodontal Disease

Study population	Age (years)	% without P.D.	Reference number
Norwegian army	19-25	0.4	38
Ontario civil servants	20-24	4.0	289
Bambay City, India	11	5.1	288
Bambay and Bassein, India	11	0.6	330
	13	0.0	
Madhya Pradesh, Central , Indian rural population	16-20	1.1	267
	26-30	0.0	
Icelanders	4+		93
Chileans	25-29	31.3	17
	30-34	24.2	
	35-39	18.8	
	40-44	11.8	
U.S. adult population, probability sample	25-34	M: 26.3	202,
		F: 37.6	212
	35-44	M: 22.1	
		F: 33.1	
U.S. urban population	20-29	52.7	358
	30-39	46.5	
	40-49	36.5	

(B) Prevalence of Gingival Abnormalities

Study population	Parameters	Age (years)	Percent	Reference number
Thailand	% with gingivitis	8-12	100.0	230
Scotland	% with gingivitis	13	99.4	282
Boston	% with gingivitis	13-15	M: 88	273
			F: 75	
Tokelau Island, N.Z. (primitive life)	% with gingivitis	10-14	M: 11.5	22
			F: 7.4	
		15-19	M: 20.0	
			F. 15.3	
		20-24	M. 48.1	
			F. 59.4	
Canada: B.C.	% with abnormal gingivae	5-13	24.7	235, 236
Sask.			19.9	
Ont.			31.3	
N.S.			48.0	
French Polynesians	% with gingivitis	4-9	M: 14.4	20
			F: 13.4	
		10-14	M: 31.1	
			F: 23.3	
		15-19	M: 33.3	
			F: 21.9	

(C) Percentage of Subjcets With One or More Periodontal Pocket

Study populations		% of subjects with pocket			Reference
		40-44 yrs	45-49 yrs		
Chilean		46.2	59.7		17
Madhya Pradesh, Central	M:	92.20	92.30		267
Indian rural population	F:	90.55	93.40		
Rural Nigeria: Yoruba		90	93		400
Ibo		61	70		
London and Warrington,	M:	97.3	99.2		401
Great Britain	F:	100.0	97.1		
		35-44 yrs	45-54 yrs		
Tokelau Island, N.Z.	M:	48.8	76.6		22
	F:	44.0	83.3		
U.S. adult population,	M:	29.7	36.9		202,
probability sample	F:	20.5	29.6		212
		40-49 yrs			
Ethiopia		47.0			
U.S. urban population		33.1			
		9 yrs	11 yrs		
Ontario children		82	73		283
		12 yrs	13 yrs	15 yrs	
Bambay and Bassein, India		0.6	2.0	9.2	330
Surrey School children,	M:	6.8	15.7	21.4	402
Kingston, England	F:	21.4	27.5	47.4	

(D) Mean Periodontal Index Scores (Russell's PI)

I. Studies made by members of a research team (N.I.D.R., U.S.A.)

Study population		Mean PI at 40-49 yrs		Reference
		Civilian only	All study population	
Chilean		2.74	2.09	17,185
Ethiopia		1.86	1.86	181,247
Alaska:	primitive Eskimo	1.17	1.45	180,367
	urban Eskimo	2.31	----	
Ecuador		1.85	1.43	183,368
South Vietnam:	Vietnamese	2.18	2.62	184,368
	Hill-tribemen	3.97	----	
Columbia		2.21	2.28	186
Thailand		3.30	3.06	187
Lebanon:	Lebanese	2.98	3.02	188
	Palestinian-refugees	3.52	----	
Burma		3.58	3.24	191
Jordan:	Jordan civilian	3.96	----	192
	Palestinian-refugees	4.41	----	
Trinidad:	East Indian	4.16	4.16	189,469
	Negro	3.72	3.72	
West Indies		----	4.36	189
Baltimore:	White	1.03	----	73
	Negro	1.99	----	
U.S. urban white population		1.34	----	358

(D) Mean Periodontal Index Scores (Russell's PI) -- Continued

II. Studies made by other observers

Study population		Mean PI		Reference
		40-49 yrs		
Hailuoto Islander,	M:	4.82		1
Finland	F:	3.02		
Osegere villager, W. Nigeria		5.80		109
Chinese Army personnel		1.26		110
Ecuador civilian and	M:	2.31		154
Montana Indian	F:	1.97		
Iran urban population		3.37		163,164
U.S. urban population	M:	1.58		358
	F:	1.26		
Ceylonese	M:	4.22		461,463
	F:	4.96		
4 ethnic grs.: Shinhalese		4.50		
Tomil		4.29		
Moor		4.08		
Others		2.63		
3 places: Anuradhapura V.		5.06		
Jaffna Village		4.36		
Colombo		3.49		
		40-44 yrs	45-49 yrs	
Ont. Dept. Highway	M:	1.92	3.33	116
employees, Canada	F:	1.76	2.92	
Melbourne, Australia	M:	3.20	3.05	243
	F:	1.64	2.59	

(D) Mean Periodontal Index Scores (Russell's PI) -- Continued

II. Studies made by other observers -- continued

Study population		Mean PI		Reference
		40-44 yrs	45-49 yrs	
Rural central India	M:	4.45	4.80	267
at Madhya Pradesh	F:	4.63	4.88	
Rural Nigeria: Yoruba		3.3	3.6	400
Ibo		1.8	3.2	
London and Warrington,	M:	4.70	5.16	401
Great Britain	F:	4.51	4.73	
		35-44 yrs	45-54 yrs	
Rural Maori, N.Z.	M:	4.75	4.86	22
	F:	1.53	3.58	
Uraguay		3.78	4.42	190
U.S. adult population:	M:	1.27	1.62	202,212
	F:	0.82	1.23	
White	M:	1.22	1.55	
	F:	0.74	1.11	
Negro	M:	1.67	2.06	
	F:	1.30	1.92	
Maori population at Tiki-TikiRangitukia		3.90	4.43	259
Israel rural settlement	M:	2.10	2.77	352
	F:	1.56	2.71	

(D) Mean Periodontal Index Scores (Russell's PI) -- Concluded

II. Studies made by other observers -- concluded

Study population			Mean PI	Age (yrs)	Reference
Norwegian Army			M: 0.80	19-25	38
Sudan			2.42	30-39	103
			M: 2.3		
			F: 2.5		
Rural Indian (Bassein)			1.53	19-30	153
Dental students in		Calcutta	1.40	21.78	201
India and Norway:		Patna	1.44	22.45	
		Lucknow	1.10	22.15	
		Bombay	1.14	24.34	
		Oslo	0.32	23.89	
Singapore:	Chinese		0.91	21.45	279
	Malays		0.70	21.41	
	Indian-Pakistan		0.86	21.40	
Ugandan:	<u>Tribe</u>	<u>District</u>			
	Bakiga	Kigezi	1.14	20-29	414
	Batoro	Toro	2.19		
	Acholi	Acholi	2.40		
	Bagisu	Mbale	2.55		

closer guess about such observers' errors. As an example, Sheiham (401) observed the periodontal disease severity in British workers in London and Warrington to be roughly three times that of the U.S. population (202,408). But we do not know how much the observer variation attributes to such high scores amoung British data. Even when comparing simpler indices such as the percentage of subjects with positive periodontal findings (212,289), or with one or more periodontal pockets (153,212,288,402), the definitions seemed to differ from one observer to another. This explains, at least partly, why Ontario civil servants (289) had such higher proportion of subjects with periodontal disease than other western population (17,202,212), and why Ontario school children (283) and Norwegian army personnel (38) had incredibly higher percentages of subjects with periodontal pockets than Indian children (330), Surrey school children (402), and French Polynesians (20).

It is quite fortunate that there have been studies (17,73, 180,181,183-189,191,247,358,367,368,469) carried out by members of a research team (Epidemiology Branch, NIDR) who had standardized their methods and criteria before they went into the field. Other data were obtained by examiners who had worked with the same research team long and closely enough so that a useful degree of comparability could be assumed (22,109,116, 153,159,163,164;190,194,243,259,267,352,400,461,463). When data from these two sources were arrayed together, a regional distribution in periodontal disease could be seen. In general, the more developed populations and regions were associated with less prevalence and severity of periodontal disease.

When different ethnic populations reside in completely different geographic regions, it is almost impossible to decide which one of these two factors, ethnic or region, is the main factor responsible for the different prevalence of periodontal disease among various ethnic groups. There are some periodontal studies, however, which were conducted among various ethnic groups living within one well defined geographic region (20,73,202,212,213,243,279,352,366,367,368,400,414,415,463,469).

A periodontal study among 9 ethnic groups in Israeli rural settlements revealed great variations, both in the severity and prevalence of periodontal disease (352). The study was not designed to explain such variations. Slome (415) also reported that post-partum Jewish women in Israel who were born in East Europe and the Near East had a higher periodontal disease prevalence than those born in West Europe, North Africa, Far East and Israel. Since age was not standardized in this comparison, the conclusion was of doubtful validity. When different severities of periodontal disease were found between Vietnamese and Vietnam hill tribes (368), between Yoruba and Ibo rural Nigerians (400), among four distinct tribes in Uganda (414), and among five ethnic groups in French Polynesia (20), corresponding differences in calculus and/or debris were also demonstrated. Wertheimer and his co-worker reported, however, that in their study in Trinidad, East Indians exhibited a slightly greater severity of periodontal disease in several age groups than the Negroes even though the OHI scores of the East Indian tended to be slightly lower than those of Negroes (469). (Transposition of the headings of Negro and East India on their table has been confirmed by authors of this study through personal communication). This unusual relationship between PI and oral deposits (in this study, materia alba), was also observed by McCombie <u>et al</u> among Chinese, Malays and Indian-Pakistanese in Singapore (279).

In the U.S.A., white persons had, on the average, much better periodontal health than the Negroes of the same age (73,202,212,366). It was also found that the Negroes presented with more materia alba (366) and higher OHI scores (213). Taking factors of age and materia alba or OHI scores into consideration simultaneously, the difference in PI scores between white and negro persons practically disappeared (213,366). Similar findings were reported by Lilienthal <u>et al</u> in their study of British-Europeans and Mediterranean-Asians in Melbourne, Australia (243). On the other hand, Waerhaug demonstrated that OHI scores and age factors failed to explain adequately the different severity in periodontal disease among four ethnic groups in Ceylon (463). This was more apparent when such comparison were made among those who belonged to high OHI categories. It suggested an interaction between OHI and other unknown factors in regard to their effect on PI scores. Waerhaug suggested that variation in protein intake among four ethnic groups might be responsible for their differences in severity of periodontal disease, although direct evidence of protein intake in relation

Populations and Regions with Relative Degrees of Prevalence and Severity of Periodontal Disease

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Low (PI: 1.03 to 1.34):

- U.S. white population
- Alaska-primitive Eskimo

Low median (PI: 1.85 to 2.62):

- U.S. negro population
- Urban Eskimo
- Canada
- Australia
- Ecuador and Columbia
- Vietnam-Vietnamese
- Ethiopia and Nigeria-Ibo
- Israel

High Median (PI: 2.74 to 3.97):

- Chile
- Trinidad-Negro
- Lebanon, Jordan civilian and Iran urban population
- Malaya, Thailand, Burma and Vietnam-Hill tribes
- Nigeria-Yoruba

High (PI: 4.10 to 5.80):

- Uruguay
- Trinidad-East Indian
- Jordan-Palestinian refugees
- Maori population, N.Z.
- India and Ceylon
- West Nigeria-Osegere villagers

to periodontal condition is still very weak.

Various ethnic groups who, though residing within a defined geographic region, often belong to different social classes and cultures. Therefore, their dietary habit, nutritional status, personal hygiene practice, professional dental care and other factors associated with social or cultural patterns may well vary from one ethnic group to another and may be responsible for the different prevalence and severity of periodontal disease among these groups.

Thus, it seems clear that there is a distinct prevalence and severity of periodontal disease among populations of various global geographic regions and among distinct ethnic groups within a defined region, and that factors associated with socio-cultural pattern may be responsible for this distribution. But the underlying determinants of disease prevalence and severity are far from understood. Oral deposits, either debris, calculus or both, sometimes, but not always, can explain part of the variation of disease distribution among various populations, but their exact role in the pathogenesis of periodontal disease is still an unanswered question which shall be discussed in detail in section I.3.6.

I.3.4. Socioeconomic factor

Socioeconomic status is a theoretical concept still awaiting clear definition. So many variables, such as years of formal education, occupation, family income, living condition, social prestige, and so on, are encompassed in the concept that in practice a single variable which can be objectively defined is commonly used as an indirect indicator for epidemiologic purposes (262).

Years of formal education, family income and occupation are the three variables which have been most commonly used as indicators of socioeconomic status in epidemiologic studies of periodontal disease. Many surveys demonstrated that better periodontal condition was associated with more years of formal education (24,38,175,212,296,366,415,463), more family income (164,212,288,463),and more favorable occupation (9,164,255,278, 296,366,401,415). Whenever periodontal conditions of military and civilian groups studied, the former group consistently had much better periodontal health than the latter group of comparable age (154,361,368,424).

Corresponding to better periodontal health among persons of higher socioeconomic status, this group also had less dental deposit, measured as debris, calculus or both (38,154,164,175, 213,255,361,463) and brushed their teeth more frequently than did the lower socioeconomic group (24). If the factors of age and dental deposits or toothbrushing frequency are held constant, the difference in periodontal severity between higher and lower socioeconomic groups dropped to negligible levels in most studies (9,213,243,368), but remained significant in adult populations in Ceylon (463) and Birmingham, Alabama (366). In his study in Ceylon, Waerhaug compared PI scores of various educational level and family income groups who belonged to the same category of age and OHI-S score, and found no difference among subjects who scored low in OHI-S, but major difference in PI still remained among subjects who had high OHI-S score (463). Thus, these results indicated an interaction between OHI scores and factors associated with these two socioeconomic variables, in regard to their actions on periodontal condition. It was not fully understood which factors interact with oral deposits on periodontal disease of Ceylonese adults, but from the fact that such an interaction phenomenon was not apparent among developed countries such as the U.S.A. (213), Norway (9) and Australia (243), malnutrition among low socioeconomic persons in Ceylon may have been the most likely factor which made periodontal tissue less resistant in these people than that of well-nourished persons, to equivalent amount of oral irritants. Russell and Ayers reported that, among adults in Birmingham, Alabama, more educated persons continued to have lower average PI scores even after age and presence or absence of materia alba were held constant (366). Since they considered only prevalence of materia alba, instead of debris and calculus, their results should not be considered as contradictory to the study made by Kelly <u>et al</u> on U.S. adult populations (213).

Although years of formal education, family income and occupation are used as indirect indicators of socioeconomic status, each variable may measure a different component of the socioeconomic complex, and it is not surprising that degree of associations between disease and socioeconomic status may differ according to which measure is used (262). Kelly and Van Kirk interpreted that family income implied the ability to purchase dental care, whereas years of formal education implied a greater awareness of what appropriate dental care was (212). Their study on U.S. adult populations showed that the correlation of PI score was higher with years of formal education than with family income, and that adjustment for years of education reduced the variance in the mean PI scores attributable to family income to roughly a quarter of the original value (212). A periodontal study on an adult population in Birmingham, Alabama revealed that, among white persons, those favorable occupations had, in general, much milder periodontal disease than those with unfavorable occupations. Data from Negroes showed the same trend, but there was little difference either in educational level or mean PI score between Negro craftsmen and foremen, operators and service workers, and common labourers which ranged from the third to the fifth occupational classes. Hence, for either race, the data suggested that years of education rather than occupational class was more closely associated with the periodontal condition (366).

Socioeconomic status which is based on a number of variables was employed in three epidemiological studies of periodontal disease (109,296,299). Mobley and Smith devised a socioeconomic index which was based on occupation, source and amount of income, education of parents, home ownership or rental, number of children in the family, crowding in the home, home facilities, types of house and dwelling area. Their study on negro children in Tennessee revealed that low periodontal disease scores tended to occur in persons in the upper status and high periodontal disease scores in those of lower status (

296). With similar criteria of socioeconomic status, Moore et al reported better gingival condition among children of higher socioeconomic status in Indiana (299). Enwonwu and Edozien studied periodontal disease among 402 Osegere Villagers and 539 Yoruba family members in Nigeria (109). These two populations differed greatly in living standard, dietary habits, nutrition and general health status such that the former group could be classified as being of a low social class and the latter group as coming from a high social class. The authors found that for each age group, Osegere Villagers had much higher mean scores of PI, DI-S, CI-S and OHI-S than Yoruba family members. Within the same categories of age group and CI-S score, Osegere Villagers had still much higher average PI score than Yoruba family members. Furthermore, the percentage of the total variance of PI scores which could explained by age, DI-S and CI-S differed markedly between these two populations, about 30% for Osegere Villagers and 81% for Yoruba family members. It, therefore, suggests that among Osegere Villagers, factors besides age and oral deposits were responsible for the greater severity of periodontal disease than that observed in Yoruba family members. The four previously mentioned social variables remained to be further examined as to their exact role in the pathogenesis of periodontal disease among poverty-stricken populations (109).

Demand for and degree of professional dental care were occasionally used as indirect indicators of socioeconomic status, because persons of greater formal education, income or occupational status received more dental care relative to their needs and made more preventive dental visits (208,209). Studies made on volunteer employees from Ontario Department of Highway (116) and post-partum Jewish women in Israel (415) revealed that severity of periodontal disease was inversely associated with the frequency of dental visits. McCombie and Stothard found that among children of Greater Vancouver, the proportion of children having gingivitis was lowest among those who had received complete treatment for dental caries, highest among treatment-neglected children and intermediate among those who never needed caries treatment or only received partial treatment for caries (420). Similar findings were reported by Russell and Ayers in their study on adult population in Birmingham, Alabama (366). The school system in Dallas classified children into accelerated and non-accelerated classes. Zimmerman and Baker reported that children of the accelerated classes had a lower average periodontal score than those of non-accelerated classes (475).

There were three studies which did not show consistent or significant association between socioeconomic status and periodontal condition (112,401,402). Sheiham reported that of male, employed persons in London and Warrington, Great Britain, those belonging to higher social status, as compared with those of lower status, had lower PI score. There was, however, no consistent pattern of severity in periodontal disease among female subjects in each age group and of the inherent weakness in the Registrar General's social classification whereby the daughter or wife of social class 1 male may be classified, for example, as social class 3 if she is employed as a clerical worker (401).

Sheiham also conducted a dental survey among Surrey school children age 11 to 17 years. He found that although children of higher social classes had less oral deposits than those from lower social classes, no consistent difference in PI score among children from various social classes was observed (402). It might be concluded from the data that children of this age group had not been sufficiently influenced by their family's social class, and that Russell's PI might not be sensitive enough, as compared with gingivitis indices, to discriminate the degree of gingivitis among children. Finestone studied the periodontal condition of 189 diabetic patients and reported no significant association between PI score and socioeconomic status (112). Since his study subjects came from low or low-middle income families, such small variation in socioeconomic status would, in theory, be of little use in describing the variation of severity in periodontal disease.

Thus, socioeconomic status which is based on years of formal education, family income, occupation or a combination of social and economic variables, has been consistently demonstrated to have an inverse association with severity of periodontal disease. Years of formal education, as compared with family income and occupational classes, is more closely associated with severity of periodontal disease. In more developed countries, most of the difference in periodontal disease variation of various socioeconomic classes can be explained by oral deposits, frequency of toothbrushing, dental awareness and professional dental care received. In less developed countries, malnutrition and other unkown factors which are associated with poor living standards seem to be more responsible than oral deposits for the more severe periodontal disease among poverty-stricken low socioeconomic persons when compared to the higher status group.

I.3.5 Urban - rural comparisons

Rural as compared to urban population tended to have higher prevalence and greater severity of periodontal disease (28,153,212, 296,330,469). The extent of the differences varied greatly from one study to another. Data from the U.S. National Health Survey showed that actual and age-sex adjusted expected periodontal disease scores did not differ greatly enough to indicate that periodontal disease was associated either with population density or with place description which included urban-rural comparison (212). Mobley and Smith reported similar findings in high school negro students in Tennessee (296). Benjamin et al studied rural children of 25 Indiana counties and observed that mean PI of county children tended, though not consistently, to increase as "percent of rural residents of the county" became greater (28). Russell analyzed the same data, but reported that this particular county variable was not associated either with presence or absence of periodontal disease or with PI scores of the periodontally involved children (359). Contrary to the small urban-rural difference in periodontal condition in the U.S.A. there was a greater difference between school children at Bombay (urban) and Bassein (rural) in India (153,330), and between rural and urban populations, age 15 to 50 years and over, in Trinidad (469). Gupta's study in

Trivandvum, India, revealed, quite surprisingly, that the mean PI for the urban population in all age groups was higher than that of the rural population except for the age groups sixty and over (159). The author did not interpret this unique phenomenon though his sampling method may have been of doubtful validity.

Corresponding to the more severe periodontal disease among rural than urban residents, the former group also scored higher OHI (153,213,469) CI and DI (153) and Plaque Indices (330).

Although urban rural populations may differ in socioeconomic status and availability of professional dental care, no studies have consedered both variables simultaneously when comparisons of prevalence and severity of periodontal disease were made between urban and rural groups. Since Russell's study on rural children in Indiana counties indicated that "median school years of rural persons 25 years or older in county" rather than "the percentage of county residents classified as rural" was associated with the PI scores of periodontally involved children (359), it is conceivable that the urban-rural difference in periodontal condition, if present, may drop to negligible levels if socioeconomic status is held constant.

It seems fair to conclude that though rural populations tended to have higher prevalence and severity of periodontal disease than their urban counterparts, such differences were in most cases not great, or negligible. Oral deposits are associated with urban-rural differences in periodontal conditions. Simultaneously consideration of socioeconomic status in future studies of urban-rural comparisons of periodontal disease is essential.

I.3.6. Occupational hazards

Injuries of the periodontal tissue which occur as a direct result of the occupational exposure to various specific agents are rather common. Most of this knowledge was, however, not obtained from specific periodontal surveys on various occupational groups, due obviously to the fact that periodontal involvement caused by occupational hazards was, in most cases, only part of the systemic manifestation.

Comprehensive studies of oral and particularly periodontal manifestations of occupational hazards were made by Schour and Sarnat (377) and by Walters and his co-workers (464). The following is a summary of periodontal manifestations of occupational disease according to specific etiologic agents - taken from the study by Schour and Sarnat (377).

The dental profession has been most familiar with the hazards of metallic intoxication, especially from lead, bismuth and mercury and their compounds, with regard to periodontal disease. These metals, in dust form, can cause distinct pigmentation, without toxic symptoms, particularly on inflamed marginal gingiva (2,167,204). In acute intoxication, acute ulcerative gingivostomatitis together with gingival pigmentation has been observed, and in cases of mercury intoxication, destruction of underlying bone has been reported (2). Since gingival pigmentation can appear in the absence of real intoxication, it has for some time been used as an indicator of chronic exposure to these metals, yet it is a very crude index because the degree of gingival pigmentation depends to a large extent on preexisting inflammation.

Other chemicals such as phosphorus (377), arsenic (178), and chromium (238) may cause necrosis of alveolar bone with loosening and exfoliation of the teeth. Inflammation and ulceration are usually associated with destruction of the underlying tissue. Benzene intoxication is accompanied by gingival bleeding and ulceration with destruction of underlying bone (377).

Most knowledge of oral manifestations of occupational hazards came from studies made during the first half of this century. Since then rapid industrial development has led to a far greater potential occupational exposure to each specific etiologic agent (121). In addition to this, remarkable advances have been made in application of organic chemicals in industrial techniques, but few or no studies have been conducted to assess these new agents as to their implication on periodontal health.

Thus, there are definite periodontal manifestations from various occupational exposures. Although early recognition and treatment of oral disease are important, the prevention of these disease should be part of a total occupational disease prevention program.

I.3.7 Nutrition, diet and systemic conditions

I.3.7.1. Nutritional factors and dietary habit

I.3.7.1.1. Population surveys

There were three studies which attempted to relate the ascorbic acid levels to periodontal condition among selected populations (317,318,388) and none of them found significant association between these two variables. For any one who tried to assess the periodontal effect of a nutrient on a population whose intake variation of that nutrient was well within normal range (388) or relatively small (317) as compared with possibly much greater variation if a population of wider social background had been studied, no association between the nutrient and periodontal score can be expected. A study by Perlitsh on personnel wintering in Antaractica (318) did not simultaneously consider other factors such as age, brushing frequency and so on, instead it only tried to correlate plasma ascorbic acid level with gingivitis in indoor and outdoor workers.

Slome reported that postpartal Israeli women with low hemoglobin levels, irrespective of their parity, had healthier gingivae than women with hemoglobin of 10% and over (415). But he explained that the anemic gingivae might have masked the inflamed gingivae by their anemic paleness. This effect might also have been due to his small study sample.

A study on dental patients by Lainson <u>et al</u> demonstrated no association of PI with either mocrohematocrit, hemoglobin or total R.B.C. count (231). This was possibly the result of selecting patients who had moderate to severe periodontal disease, instead of including subjects with a wider span of disease, thus, although three-fifths of the female subjects had a low microhematocrit level, still no association between microhematocrit and periodontal score was found.

A periodontal survey by Emslie among children and young adults in Sudan and Nigeria (105) revealed that neither acute ulcerative gingivitis among Nigerian nor periodontal score among Sudanese correlated with protein malnutrition. In Sudan, girls at age of 10 to 19 years had a much lower hemoglobin level than boys, but the former also had a lower average PI for each age than the latter. It is difficult to interpret this finding because growth differs greatly between sexes at this age as do other factors such as tooth cleansing.

Enwonwu and Edozier studied children and young adults in Western Nigeria (109). They found that among subjects of the same age and calculus score, the group PI score was higher among the low S-E group than in the high S-E group. Children from low S-E classes had lower serum albumin, higher globulin, lower ratio of albumin to globulin, and lower hemoglobin levels than did children from high S-E classes. Protein malnutrition, therefore, might be responsible for the progression of periodontal disease among children from low S-E classes.

In his study in Ceylon, Waerhaug reported that vitamin B deficiency, as diagnosed clinically, was associated with higher PI score, and this association exaggerated when the OHI score increased, but nearly disappeared when the OHI score was very low. While the difference in PI of each age between well nourished and malnourished males was not apparent, such difference among females was more pro[illegible]ced (463). All these facts pointed out an interactive relation between nutritional factor and

OHI score as well as between nutritional factors and frequency of child-bearing in women living under primitive conditions, in terms of their effect on the progression of periodontal disease.

There have been many dental surveys carried out in connection with the nutritional studies sponsored by the Interdepartmental Committee on Nutrition for National Defense (I.C.N.N.D.) which have covered many geographic areas throught the world. The most common biochemical tests carried out were total serum protein, hemoglobin level, serum ascorbic acid, serum vitamin A, urinary excretion of thiamin, riboflavin and N' methylnicotinamide. A study by Littleton in Ethiopia showed that for similar age groups, those with low or deficient vitamin A or C levels had significantly higher group PI scores and G.R. rates (247). Other studies which had **assessed** a simple linear correlation co-efficient between PI or G.R. and each biochemical data indicated occasional significance (17,368). The magnitute of associations was not great and could happen by chance from the large number of significant tests in a single study. Furthermore, part of these associations diminished when partial correlation co-efficients were tested in which factors of age and OHI score were held constant (18,247,362,368). In one article the authors made following statement: "less than 10% of the variance in group PI scores remain to be explained after the combined influence of age and mouth cleanliness (debris and calculus) has been estimated. A residual factor wholly independent of age or hygiene (debris and calculus), therefore, can have little effect on periodontal disease as scored by PI." (362).

Some comments have to be made about these population surveys before meaningful conclusions can be drawn. 1) when a population survey was conducted in a geographic area where the population nutritional status was homogenous, ie. very poor in all foregoing studies, because of the small intake variation of each nutrition, little or no association between nutritional state and periodontal score could be expected. 2) Since some biochemical data also were associated with age and OHI score (18,368), partial correlation coefficients between biochemical data and periodontal scores with age and OHI standardized would certainly lead to less correlation than their simple correlation coefficient. 3) Since we do not yet understand what role ---- cause, result or both? ---- which subgingival calculus plays in the pathogenesis of periodontal disease, standardization of this variable when correlating nutritional factors and periodontal scores, therefore, becomes a dubious procedure. 4) No one knows for certain what role age actually plays in the disease process either, but a logical guess is that it mainly indicates, indirectly, the duration of existing periodontal disease and etiological factors. Age, therefore, may be an important factor in disease progress rather than disease initiation. Thus, when a multiple correlation coefficient for a nutritional factor and age over a rather wide range with periodontal severity is estimated, age will almost completely dominate such correlation. 5) Biochemical data indicate nutritional status immediately preceding the tests, whereas periodontal disease is a longstanding chronic disease; a great deal of caution is therefore required in interpreting the association of these two variables in a cross-sectional study.

With these considerations, it is easier to understand why, from studies in Chile and Ethiopia, the combined effect of age, sex, oral debris, supragingival calculus, all biochemical data and molar attrition score explained only one-third and one-half respectively of the total variance of PI and GR (18,247),whereas from studies in Lebanon and South Vietnam (362), the combination of age and OHI (including subgingival calculus in addition to oral debris and supragingival calculus) explained nine-tenths of the total variance of group PI score. In their study on Eskimo national guards in Alaska (367), Russell et al found that although their study population had high prevalence of gingivitis, it had relatively little loss of alveolar bone. They also reported that supragingival calculus was not significantly associted with alveolar bone loss, and suggested that high protein intake might have prevented alveolar bone resorption. A study by Enwonwu and Edozien in Western Nigeria (109) also revealed the combined effect of age and OHI explained four-fifths of the total variance of PI among subjects from higher social class (Yoruba family members), whereas it accounted for only three-tenths of the variation among those belonging to low social class (Osegere Villagers). Thus, great difference in hemoglobin and other serum protein levels between the high and low social group in this study might be the most important factor responsible for much greater periodontal severity in the low social group (Osegere Villagers).

To summarize, most population surveys did not find that the deficiency of any specific or group of nutrients, as determined by biochemical analyses, was significantly associated with periodontal severity. These negative findings might result from rather homogenous nutritional state and wide age range of the study populations. Other studies suggested that protein malnutrition might be responsible for greater periodontal severity among poverty-stricken people as compared with better nourished groups dwelling within the same geographic area.

I.3.7.1.2 Clinical Trials

Although clinicians have long been impressed by the detrimental effects of nutritional deficiency on periodontal health, the delayed appearance of population data and the general lack of application of epidemiology and statistics in clinical trials by the dental profession have greatly handicapped extensive use of this method in periodontal research. Lacking a solid scientific grounding, many periodontal studies are open to criticism.

A) Carbohydrates

Choosing systemically healthy dental students of a study, Cheraskin, Ringsdorf and their associates initially scored on gingivitis (61), measured sulcus depth (55,57) and estimated clinical tooth mobility (56,59). The students were then divided randomly (arbitrary?) into an experimental and a control group. During three experimental days, the former group drank either sucrose (55,56,61) or glucose (57,59,61) solutions and the latter group consumed artificial sweet drinks. On the fourth day, the parameters were estimated again by the same examiners who were supposed to have no knowledge of either the kinds of drink or the initial scores of each individual. They reported that while no significant changes in mean scores of parameters secured among the control group, the experimental group showed a significant score increase in all three parameters studies. From their data however, it became obvious that: 1) random division into experimental and control group sometimes resulted in quite different initial mean scores (56); 2) the differences in parameter scores either between experimental and control group at the end of the experiment or between the initial and the final mean scores within each group were so small that they could have occurred well within observers'error; and 3) the surprising consistency in a very high proportion of experimental subjects to have increased scores of all studied parameters with no change for the remainders while a very low proportion of control subjects had increased parameters' scores, casted doubt as to whether planned blind assessment actually worked out at all.

Thus, the effects of sweet drink on gingivitis, sulcus depth or clinical tooth mobility are still not very clear. Better study design such as matching for pairs between two groups, larger sample size and longer experimental period, as well as more appropriate methods for data analyses are essential for future studies.

B) Protein

Cheraskin, Ringsdorf and their co-workers studied the effect of protein supplementation against placebo supplementation on the periodontal disease of dental students (49,50,51,339, 342), and adult personnel of the Medical Center and Fire Department in Alabama, U.S.A. (60,62). Initial biochemical examination of serum protein, albumin and globulin levels indicated all subjects to be within normal range. Following initial estimation of the gingivitis score (50,60,339), sulcus depth (51,342) and tooth mobility (49,62), the study subjects were randomly (arbitrary?) divided into an experimental and a placebo group. The former group received daily protein supplements which were either a mixture of essential amino acid (339, 342) or animal protein (49,50,51,60,62). Each experiment ran for either three days among dental students or two weeks among adult personnel of the Medical Center and Fire Department. At the end of each experiment, the same observer estimated the condition of the same parameter with no supposed knowledge of the kind supplements or initial score of each individual. Results showed that while there was significant improvement in the condition of periodontal disease among experimental subjects, little changes occurred among placebo group. From their study of sweet drink **same questions could be asked here too.**

With a sort of cross-control design, Lederman and Hazen studied the effect of protein supplements on gingival disease among young adults (233). Pairs of experimental and control subject were matched according to initial gingivitis score. The authors reported a beneficial, though transcient, effect of protein supplements on gingivitis severity among subjects who started with protein supplementation during the first three weeks and changed to the placebo during the next weeks. No initial nutrient data or other quantitative data were reported.

It was rather difficult to accept the conclusion that severity of periodontal disease among those young adults could be improved by supplements when there was no evidence of protein deficiency to begin with. Until data from more critical studies are available, no conclusion is immune to serious questioning.

C) Vitamin C

Vitamin C in pure form as well as in natural fruits such as orange and grapefruit has been used as a supplement in many clinical trials.

A clinical trial on subjects whose initial levels of ascorbic acid in plasma and tissue was relatively low, indicated that vitamin C supplements reduced the severity of gingivitis (99,229,260,281,346), sulcus depth (100) and tooth mobility in the experimental group (101). Parfitt and Hand made a similar study on inmates of a mental institute who initially had very low plasma levels of ascorbic acid, but they did not find any significant reduction in severity of gingivitis among the supplemented subjects (317). The authors also reported that although the experimental subjects had received 500 mg of vitamin C daily for a total of six weeks, their ascorbic acid in plasma did not reach to normal levels by the end of the experiment.

Studies on subjects who initially had very high (232,319) or optimal levels (141) of plasma ascorbic acid showed no reduction in gingivitis score following dietary supplementation of vitamin C. Glickman and Dines showed that by the end of the experiment the ascorbic acid levels in plasma of the supplemented subjects increased, while little change occurred in the tissue level (141). They also reported that subjects whose gingivae were treated immediately following initial assessment of gingivitis score had significantly better gingival health than others at the end of the study. Results from this study (141) as well as from others (99,100,101) which had considered effects of both supplementation and initial prophylaxis, indicated that co-existing factors, both local and systemic, were responsible for progression of the disease.

Thomas et al studied gingival hue (443) and resorption of alveolar bone (442) among dental students. They not only supplemented the diet with orange juice of experimental subjects but also requested control subjects to eliminate citrus fruits from their diets throughout the study period of one year. The results showed a marked reduction in scores of gingival hue among the experimental subjects and increase in severity of gingival hue among control persons. But there was no significant difference in the height of alveolar bone between the two groups at the end of the study, although the average rate of decline of alveolar bone height suggested slightly slower alveolar bone resorption among experimental students than control ones. It is questionable that a study lasting only one year and lacking a reproducible method for measuring height of alveolar bone could yield significant information in this area.

Roth (354) and Carvel (47) reported a reduction in the severity of gingivitis following dietary supplementation with bioflavonoid and vitamin C. Since their study subjects were clinical patients with varying ages, were small in number and provided no information about the level of vitamin C in plasma or tissue, only limited conclusions can be drawn from these two studies.

Four other studies demonstrated no significant reduction in the severity of periodontal disease following supplementation with vitamin C (75,227,311,427). None of them assayed either initial or final ascorbic acid level in the plasma of study subjects. For a study population like the young adult personnel in the Royal Air Force (427), it seems unlikely that a severe deficiency of vitamin C could have existed. In Kutscher's study on dental patients, the initial scoring of gingivitis was done according to the gingival bleeding and sensitivity by subgingival curretage over one-half of each mouth, while the final scores were made in like fashion on the previously unscaled half. Thus, initial and final score did not represent the same tissue area (227). All children and adolescents studied by Coven, received an initial thorough whole mouth prophylaxis. The experiment lasted only 28 days. Marked improvement in the gingival health of both experimental and placebo groups, without a significant difference in the degree of improvement, indicated that beneficial effects from initial prophylaxis overruled supplementary effects in such a short study period (75). O'Leary <u>et al</u> employed very small samples, four or five subjects in each of four groups, whose age also varied greatly, hence, their negative results should not be interpreted as showing no association between vitamin C and gingival disease.

In summary, vitamin C supplements could not reduce the severity of periodontal disease in subjects who had no evidence of a vitamin C deficiency. For subjects who had an apparent deficiency in this vitamin, supplementation with vitamin C in its pure form or in natural fruits might, to some extent, improve gingival health and reduce sulcus depth. Its effect on alveolar bone resorption is not certain. Considering the fact that there were only two studies in which the control subjects were asked to eliminate citrus fruits from their diets, and that vitamin C deficiency was likely to be only a minor factor of many responsible for the severity of periodontal disease and that most foregoing trials did not consider simultaneous other factors, especially tooth cleansing, it should not be too surprising to find that the effects of vitamin C supplementation varied greatly from one study to another.

D) Multivitamin supplements

The beneficial effects of dietary multivitamin supplements on gingival health and tooth mobility were reported to be slightly masked by the destructive effects of orthodontic banding (96). Ringsdorf and Cheraskin supplemented dental students with multivitamin- trace mineral for three days and found a significant reduction in gingival inflammation (340) and sulcus depth (341). Since there was no evidence of vitamin deficiency among those students, and the differences in periodontal parameters between experimental and placebo groups as well as between initial and final scores of the same groups were not great and could occur from observer error, their results could be taken only as a motivation for more critical studies.

Keller et al reported no reduction in gingivitis score on hemiarches of subjects who had received multivitamins for three weeks (210). This result was interesting because these subjects started with moderately low levels of ascorbic acid in the plasma and demonstrated increased levels at the end of the experiment. The ascorbic acid levels in tissue were not tested. Although Dachi et al found no reduction in sulcus depth on experimental subjects supplemented with multivitamins for five days (78), this may have been because the study subjects, dental students, had initially very shallow gingival sulci (only 10% of them had sulci deeper than 2 mm), therefore any possible beneficial effect on sulcus depth could have been masked by observer error. Comparing the effects of a placebo, a vitamin B complex alone or together with vitamin A, D or C on the reduction of gingival inflammation among children and young adults, Coven reported that the best results came from the subjects who had received a combination of vitamin B complex, A, D and C (75).

Finally, to compare effects on periodontal parameters between regular and sustained -release multivitamin supplements, Cheraskin and Ringsdorf conducted a four day experiment on fifty dental students whose initial vitamin levels were not stated. Half of the subjects received the sustained - release formula, whild the remainder took the regular - release type. Results showed that while both groups improved in periodontal health, subjects who had received the sustained - release formula, as compared with those of the regular -release formula, had greater reduction in gingivitis scores (53), sulcus depth and tooth mobility (54). In a separate report from this study, Rechards _et al_ reported a higher average ascorbic acid level in the tissue at the end of the experiment among the "sustained" as compared to the "regular" group (336). Since no placebo group was included in this study design, the exact degree of improvement in periodontal health from these two kinds of supplements is an open question. Also from the data, it was obvious that the differences between the two groups in sulcus depth and tooth mobility was not great (54).

Same comments and conclusions about vitamin C supplement are applicable here. None of the studies mentioned above attempted to standardize other disturbing factors when comparisons were made between multivitamin and placebo supplements in terms of effects on periodontal disease. Thus, supplementary effects might have been over - or under-estimated. Until data from more critical studies become available, the best conclusion one can make is that multivitamin supplements tend to reduce gingival inflammation and that their effect on sulcus depth or tooth mobility is not certain.

E) Summary from clinical trials

Results from the foregoing clinical trials did not greatly substantiate or clarify the nature of associations between nutrients and the severity of periodontal disease as found in population surveys. While the results from many clinical trials indicated that periodontal disease tended to be reduced in severity by withdrawal of sweet drinks, and by supplementation of protein, vitamin C as well as multivitamins, their validity was quite questionable due to: 1) no evidence of deficiency in specific nutrient among most study subjects, 2) the possible failure in blind assessment, 3) the relatively short study periods, 4) no control over diets in placebo groups, 5) the failure to control other disturbing variables in the study, and 6) the incorrect methods of data analyses. The only finding which seemed to occur more or less consistently throughout this review was the reduction of gingival inflammation following vitamin C supplementation in subjects who initially had apparent vitamin C deficiency. That does not exclude the possibility of periodontal disease due to a deficiency of other nutrients. Instead, it means that current data do not allow many definite conclusion.

In spite of all these drawbacks, clinical trials have demonstrated the fact that nutrient factors are not major variables and certainly not the only ones responsible for severity of the periodontal disease.

I.3.7.1.3 Fluoride in drinking water --- a specific nutrient

There were nine population surveys which investigated the association between fluoride concentration in drinking water and periodontal disease. Four of them were made on children or adolescents and five others on adult populations.

Brown and his associate (40) assessed the prevalence and severity of gingivitis among school children of age six to fourteen years in Brantford, Sarnia and Stratford, Ontario. The disease was found to be equivalent in Brantford, where drinking water had been artificially fluoridated for 8 years, and in Sarnia where water was fluoride free. In Stratford, where drinking water contained 1.3 ppm natural fluoride, the prevalence and severity of gingivitis were much worse than in the other two communities. This was explained by the authors as a reflection of public carelessness in tooth cleansing attributable to a low caries rate of long standing. Quite similar results were reported by Moore et al (299) who compared prevalence and severity of gingivitis among 1123 white children, aged 7 to 12 years in Bloomington, Frankfort and Indianapolis, Indiana, the water of which contained fluoride ranging in concentration from 0.1 to 1.1 ppm. Ast and Schlesinger (11) studied 438 children, aged 6 to 16 years, in Newbourgh where water had been fluoridated for 10 years, and 612 children of the same ages in Kingston where there was a very low fluoride level in the water. Slightly, but significantly more gingivitis was observed among latter children. Russell (360) surveyed thses same populations, but included only those whose ages were between seven to fourteen years and ascertained the PI instead of the P.M.A. He drew similar conclusion for each of three age groups, but added that the difference in PI between two communities was due wholly to the higher prevalence of disease in Kingston. When present, disease was of about equal in the two communities. Englander and White (107) investigated the pervalence and severity of periodontal disease in 838 white native life-time residents, aged 13 to 17 years, in Aurora, water of which contained 1.2 ppm of natural fluoride and in 823 teenagers of similar nature in Rockford, Ill., which ~~had~~ only 0.1 ppm of fluoride in water. Study subjects in both cities were distributed similarly according to age, sex, socioeconomic, educational levels, health and quality of dental care. No salient differences were found in either the PI or the OHI scores between teen-agers of these two cities.

Zimmerman and his co-workers (476) studied periodontal disease on older persons in Bartlett, the water of which, until the recent defluoridation program contained 8 ppm of natural fluoride, and Cameron, Texas which had only 0.4 ppm of fluoride in water. Little differences in gingivitis or alveolar bone resorption, as assessed by the PMA and X-ray respectively were found between two groups examined. These populations were later studied by Russell (360) who assessed the periodontal disease according to percentages with pockets, mean PI scores and percentage with edentulous arches. He reported the two groups to have no significant differences in overall disease severity. Russell (359,360) compared periodontal disease between 379 subjects aged 20 to 40 years in Colorado Springs, the water of which contained 2.5 ppm of natural fluoride and 144 persons of similar ages in Boulder, Colorado, which had practically no fluoride in the water. For each of five age groups, subjects in Colorado Springs had consistently higher percentages of negative periodontal involvement, lower percentages of subjects with pockets and lower group PI scores. Once initiated, however, the disease was equally sever in the two communities. The observed differences were, therefore, wholly due to higher prevalence of disease in the community using the fluoride-free water. These findings were more interesting when taking the fact that the study subjects from Boulder had, in general, more years of education than those from Colorado Springs (359). In addition to their study on teenagers in Aurora and Rockford (107), Englander et al (106) also investigated the periodontal disease among adult populations in these two communities. Their results showed that, for each age group, mean PI scores and percentages of subjects with pockets were consistently lower among residents of the naturally fluoridated city, but subjects in both cities had similar OHI score. Barros and Witkop (17) reported that while fluoride concentrations in the water varied a great deal geographically in Chile, no such variation in periodontal condition were found.

It is obvious that none of the aforementioned studies was conducted to assess the periodontal disease blindly, and it is really a question of how much observer bias due, for example, to the emotional favouring of fluoridation, affected the study results. It seems, therefore, appropriate to mention the study by Russell (360), in which he compared the periodontal conditions between the natives and migrants in fluoridated communities. The native school children in series of communities, each with about 1.0 ppm of fluoride in its domestic water, were compared with migrant children in the same communities. There was a weak tendency for the periodontal health of native children to be better relative to that of migrants, depending on how long the latter had lived in the fluoridated community. This was, nevertheless, a very crude test because the whole community served as a unit for comparison.

It is fair to conclude from these studies that most of the differences in periodontal conditions between communities having high and low fluoride concentrations in domestic water were not great if they existed at all. There is, therefore, no adequate evidence to support the hypothesis that the use of fluoridated water results in improved periodontal health. The findings are however, wholly incompatible with any hypothesis that the periodontal tissues are harmed by use of fluoridated domestic water.

I.3.7.2 Organic systemic disease

I.3.7.2.1 Diabetes mellitus

Rudy and Cohen reported that of 581 diabetes treated in a hospital, 219 or 37.7% were edentulous, and that of 18 diabetics aged from 6 to 30 years, only one had periodontosis (355). Studies by Sheppard on diabetic patients revealed that diabetic duration and severity were positively associated with the degree of alveolar bone resorption assessed from radiographs (404,405). Considering the rather wide age range and the small number of study subjects, the association is only suggestive. None of the aforementioned studies had control groups for comparison.

There were studies which demonstrated significant differences in severity of periodontal disease between diabetic and control subjects (6,25,65,112,136,257,335). In his study on 200 hospitalized patients with ten kinds of disease categories, Arif (6) found that diabetics as well as patients with neoplasms or gastrointestinal disease, had a higher mean pocket depth than did other patient. But in this study, diabetics were on the average, older and had more oral deposits than did persons with other diseases.

Belting et al studied poorly controlled diabetic patients and found that for the age groups 30 to 39 years and 40-49 years, scores of periodontal disease were significantly higher among diabetics than controls (25). Taking all ages together and standardizing for toothbrushing frequency and amounts of calculus, the diabetics, in general, had more severe periodontal disease than the controls. Similar results were reported by Fineston and Boorujy (112) and by Ray and Orban (335). By comparing between 51 patients visiting a hospital for other illness, Glavind et al reported that for the age groups of 21 to 30 and 31 to 40 years, more resorption of alveolar bone and greater epithelial detachment were observed among diabetics than the controls, but a statistical significant difference occurred in epithelial detachment of the older age group (136). Lovestedt and Austin observed periodontoclasia in diabetics who were slightly older than controls and reported that such slight difference in age could not explain the greater proportion of diabetics with severe periodontoclasia and edentulous (257). Cohen et al conducted a three year longitudinal study on 21 female diabetic patients and 18 females without any metabolic disease (65). The study subjects ranged in age from 18 to 35 years. Annual periodontal disease examination revealed that the diabetics had consistently higher mean gingivitis scores and PI scores, but had equivalent or less amounts of soft and hard deposits than did controls. Thus rapid progress in severity of periodontal disease among diabetic patients could not be explained by coressponding increases in oral deposits.

Of six studies which did not show significant difference in periodontal parameters between diabetic subjects and control groups, five studies investigated diabetic patients whose diabetes was either very mild (30,177,285) or was already under perfect control (136,261). The study sample derived from probability sampling of U.S. population, included only a few diabetic subjects, thus, although male diabetics had, on the average, higher PI scores than age-adjusted expected scores, the difference was not statistically significant (211). Diabetic subjects studied by Glarind and his associates (136) were already under control and did not have a higher group plaque score, gingival score or deeper pockets than did their controls. But the diabetics had greater epithelial detachment and more resorption of alveolar bone than the controls which might indicate either that resorption of alveolar bone had taken place before diabetes was under control or that controlled diabetes affected only hard periodontal tissue, ie. alveolar bone, and gingival tissue was more associated with local deposits. Since Hove and Stallard employed routine dental patients as control subjects, any possible slight increase in severity of periodontal disease among very mild diabetic subjects, therefore, could not easily have been observed (177).

Although there were three studies which indicated significant association between duration of diabetes and severity of periodontal disease (112,136,404), such association might have resulted from the different ages between diabetics of longer and shorter duration. Another study failed to show association (177). There were four studies which revealed no association between severity of periodontal disease and severity of diabetes (25,112,136,177). Although Sheppard reported a positive association between insulin doses and resorption of alveolar bone (404), the small number of his study subjects and the great variation in age limited any clear-cut interpretation. Glavind et al also observed that diabetics whose retinal change was obvious, had, on the average, greater epithelial detachment than did the controls (136), but age also differed very much between two groups.

There were three histological studies on the gingival tissue of diabetics and non-diabetics and all revealed that diabetic gingivae had a much greater vascular change than did those without diabetes or hypertension (285,360.425), but there was no relation between the degree of vascular changes and severity of gingivitis (285). No characteristic change of periodontal disease with diabetes has yet been observed (261,335,405).

To summerize, poorly controlled diabetics had more severe gingivitis, deeper pocket, more loss of alveolar bone and greater epithelial detachment than did controls. For persons with mild diabetes, there was not enough evidence that they had more severe periodontal disease than persons without diabetes. Persons whose diabetes was well under control had no greater severity of gingivitis than did control subjects, but might have lost more alveolar bone which had taken place, most likely, before diabetes was controlled. Although gingivae from diabetics as compared with those from non-diabetic or non-hypertensive subjects had more vascular changes, such changes were not associated with severity of gingivitis. Finally, there was no characteristic change or periodontal disease associated with diabetes mellitus.

I.3.7.2.2 Glucose tolerance

In some studies, persons with varying severity of periodontal disease were examined for glucose tolerance and the correlation between these two variables was studied. Glucose tolerance was separated from diabetes mellitus in preceeding section because of the fact that glucose tolerance was not a factor which decided the selection of study subjects and that except for very bad glucose tolerance, glucose tolerance was not necessarily related with diabetes mellitus.

Ostrom and his associates tested fasting blood glucose level and glucose tolerance on 41 subjects with chronic periodontal disease. They found that although there was no difference in group glucose levels between subjects with chronic periodontal disease and controls, a trend existed in which the overall curve of the glucose tolerance test was higher among former than latter group of comparable age (315). The small number of the study subjects in each age group suggested a need for expansion of the study. A study by Moller and Cheraskin revealed a parabolic relationship between interval scores of oral signs, ie. tooth mobility and/or missing teeth, and glucose levels. In other word, persons with hypo- or hyperglycemia, as compared with those with normoglycemia, had higher scores of the oral sign (298). Such parabolic relation also prevailed when only subjects within small age ranges were considered. Since their material showed an extremely high coefficient of variation of glucose levels, the results obtained by distribution-bound test methods would have distorted a great deal of true association between glucose tolerance and severity of periodontal disease. Data of a similar nature but with more obvious statistical weakness were presented by Cheraskin and Ringsdorf.(52). They tested two groups of dental patients, one with and one without gingival pathosis, for either classic or cortisone glucose tolerance and reported that only the one- and two- hour determinations for cortisone G.T.T. showed a significant difference in group glucose levels between the subjects. The group with gingival pathosis had much greater variation in the glucose levels than did the group without. Looking at their data, however, one would realize that differences in both means and variances of glucose levels between two groups arose solely from data of one subject who had gingival pathosis and whose glucose levels were more than twice that of the other patients. There was no apparent difference in levels or distribution between the subjects with gingival pathosis and those without the pathosis. O'Leary <u>et al</u> reported no difference between periodontal patients and control subjects with respect to glucose levels at any point of testing time with oral or intravenous method of G.T.T. (313). All studies conducted by Shannon and his associates on new army recruits failed to show any association between glucose tolerance tested in various ways and parameter of periodontal pathosis (219,389,390,391,392,393). Since their study subjects were young (17 to 22 years of age), systemically healthy, able to take unlimited minitary duty and within normal glucose tolerance, these results could only be interpreted as applicable to a population similar to theirs; glucose tolerance had no predictive value in the severity of periodontal disease. Kelly and Engel also studied glucose tolerance on the subjects derived from the probability sampling of the U.S. adult population (211). They reported that male subjects whose blood glucose levels were above 208 mg% had higher group PI score than the age-adjusted expected value, but the excess was not quite statistically significant. Summers and Oberman studied 324 subjects 20 years or older in age, which consisted of a 10% probability sample of the Tecumseh Health Study. They reported a significant correlation coefficient between serum glucose levels and Periodontal Disease Index (PDI) scores (430). But a partial correlation coefficient, holding constant the age and cigarette consumption, indicated that the original correlation diminished to a non-significant level.

As an example of how different results could occur in studies on the association between glucose tolerance and severity of periodontal disease, it would be worth to mentioning the study by Tuckman <u>et al</u> (447). The study subjects consisted of 41 routine dental patients seeking oral diagnosis as well as 13 dental students. The authors first selected 2 groups of 12 subjects from the 41 dental patients, one with the highest and one with the lowest PI scores, and found no significant difference in average glucose tolerance between the two groups. They than selected the subjects in the same fashion but from all 54 subjects. All 12 subjects with the highest PI scores came from the 41 dental patients and all but one of 12 subjects with lowest PI scores came from the 12 dental student. There was significant difference in average glucose tolerance between these two groups. Since age differed markedly between the two groups, an analysis of covariance was done to adjust for age differences. The result showed that the difference in average glucose tolerance between two groups remained significant.

Thus, except for subjects with clinical diabetes, glucose does not correlate strongly with the severity of periodontal disease. Glucose tolerance is not the only or major factor associated with progression of periodontal disease. Therefore, when studying a population whose glucose tolerance is within normal range and/or who had relatively mild periodontal disease. no significant association between these two variables can be expected. Instead, one should study a population who, preferably, had glucose tolerance along the whole possible scale or, less desirably, had wide range of severity of periodontal disease. Only then may the association between these two variables be shown.

I.3.7.2.3 Mental retardation and related birth defects

All studies on mongoloid subjects showed a high prevalence and severity of periodontal disease (41,221,284,439). Mongols, studied by Brown and Cunningham, suffered from severe destruction of periodontal tissue which had little relation to local deposits. Calculus formation was uncommon and there was correlation between the amount of oral deposits and the severity of periodontal disease (41). Studies conducted by Kisling and Krebs on young adult mongols (221) and by Sznajder et al on mongoloid children and adolescents (439) demonstrated, however, a significant correlation between the amount of calculus deposition, but not plaque, and the severity of periodontal disease. But the proportion of variation in the severity of periodontal disease which could be explained by calculus amount was rather small. McMillan and his co-workers also observed high proportions of missing teeth and even edentulous arches in the mongoloid adults (284). Although none of the aforementioned studies had control groups for comparisons, the high prevalence, severity and characteristics of periodontal disease were remarkable enough to suggest a close relation between mongolism itself and periodontal disease.

When mongoloid and non-mongoloid mentally defective subjects of similar age and habitat were compared, the former always had higher prevalence and severity of periodontal disease and greater loss of alveolar bone (70, 203, 437). In one study which had also mentally normal subjects for comparisons, the author reported that while resorption of alveolar bone occurred in 96% and 40% respectively among mongoloid and non-mongoloid mentally defective subjects, it occurred in 6% only among control subjects (70). Johnson and Young also observed that periodontal disease in non-mongoloid mentally defective children was characterized by chronic inflammatory periodontitis leading to slight resorption of alveolar bone. In mongols, however, the pattern and progress suggested a form of Vincent's infection and periodontitis complex superimposed on an acute ulcerative gingivitis (203). Swallow reported that although periodontal disease was much more severe among mongoloid than non-mongoloid mentally defective patients of similar age, no difference between two groups in oral debris or calculus could be found (437).

When periodontal disease of mentally retarded children (43), mongoloid or non-mongoloid mentally defective children (437) was compared with other normal subjects, the formers always presented higher prevalence and severity of the disease. But a correspondingly higher prevalence of "inadequate cleanliness" was also observed among the former.

The above studies suggested that mongols had very, low resistance to progressive periodontal disease and this was not attributable to poor nutritional status (70).

The rather high prevalence and severity of periodontal disease was only partially explained by the presence of oral deposits (41,203,221,437,439). As the degree of mental retardation increased, the proportion of variation in severity of periodontal disease which could be accounted for by the amount of oral deposits decreased (70,437).

I.3.7.2.4 Cerebral palsy

All studies on cerebral palsy children revealed a high prevalence and severity of gingivitis than in non-C.P. controls (113,263, 465,468). Watson reported that C.P. children had only slightly higher prevalence of gingivitis than children without C.P. (465). Weisman found that C.P. children had much higher prevalence of marginal gingivitis than expected, that 90% of the C.P. children had one or more teeth with a pocket depth greater than 3 mm (468). Although he did not include non-C.P. children for comparisons, these findings were impressive. Magnusson and De Vol compared gingival disease between 76 C.P. children and an equal number of controls each of whom was matched with a C.P. subject for sex and date of birth. The authors reported that the prevalence of gingivitis in C.P. children was about three times that of the controls. About half of the C.P. subjects with gingivitis presented gingival hyperplasia, possibly, due to dilantin treatment. Among C.P. subjects, the prevalence of gingivitis was not associated with type of C.P., intelligence, degree of handicap, mouth breathing habit or bruxism. Although no apparent difference in frequency of toothbrushing between C.P. and control subjects existed, the former tended to have more abundant soft and hard deposits on tooth surfaces (263). Fishman <u>et al</u> also observed significantly higher group PI score among C.P. children than among other non-C.P. siblings at each of four age groups. No significant difference in OHI-S score could be found between C.P. and non-C.P. siblings, though there was a tendency for older C.P. children to have higher OHI-S score than corresponding controls (113).

Thus, it seems fair to conclude that C.P. children in general had higher prevalence and severity of gingival disease than is general observed in non-C.P. children. Such excess could not be entirely explained by relatively more oral deposits in C.P. children. Sznajder and Feniak, however, reported that systemic influence of C.P. itself on gingival disease was less than that of mongolism (440).

I.3.7.2.5 Cardiovascular disease

In a study on 1299 white, male, hospitalized veterans, Sandler and Stahl reported that alveolar resorption tended to be greater in subjects with cardiovascular disease than in comparably aged patients with other illness (371). Their later study on a similar population revealed that the percentage of patients with chronic periodontal disease was higher, but not significantly so, in patients with arteriosclerosis than in "other" patients at each age group (373). MacKenzie et al however, observed no significant difference in resorption of alveolar bone between arteriosclerotic subjects and their age-matched control (261).

Summers and Oberman studied a probability sample of 324 adults in Tecumseh, Michigan, and found that both systolic and diastolic blood pressure levels correlated significantly with Ramfjord's PDI scores in femal subjects. Correlation coefficient of systolic blood pressure level with PDI scores remained significant even after age and cigarette consumption were held constant (430). However, the proportion of PDI variance which was accounted for by systolic blood pressure level was quite small (about 7%). In separate paper, the authors also reported that the prevalence of coronary heart disease was more than twice as great in the edentulous persons (6 of 84 or 7%), as compared with the dentulous persons (9 of 324 or 3%). When the percentages were adjusted for age, however, there was no significant difference in the rate between the edentulous (4.5%) and dentulous (2.8%) persons (431).

The most comprehensive study on a properly representative population with regard to the association between cardiovascular disease and severity of periodontal disease was conducted by Kelly and Engel on probability sample of U.S. adults. The authors' findings were summarized as follows: 1) Women with hypertension had a statistically significant elevation of their periodontal scores,and hypertensive men had a near-significant elevation. 2) Periodontal scores of men and women rose with increasing systolic and diastolic blood pressure. 3) People whose peripheral pulse could not be palpated had more periodontal disease than those with palpable pulses. The elevation in periodontal scores in men was statistically significant. 4) Men and women with increased retinal light reflex had higher, than expected, periodontal scores. The elevation of scores associated with increased light reflex was statistically significant. 5) People with definite coronary heart or with hypertensive heart disease had relatively high periodontal scores. 6) People with abnormal ECG tracing generally tended to have higher periodontal scores than those with normal tracings.

Thus with currently available data, there is sufficient evidence to indicate that severity of periodontal disease was positively associated with systolic blood pressure in females, peripheral arterial disease in men and with retinal vascular changes in both men and women. Relation between periodontal disease and coronary or hypertensive heart disease was less convincing though a similar positive correlation was occasionally observed.

I.3.7.2.6 Hematologic disease

Some hematologic disorders manifest in periodontal tissue as a combination of both specific phenomenon from hematologic disease and non-specific inflammatory reaction to local irritation.

According to studies by Robinson and Sarnat, a majority of patients with sickle-cell anemia exhibit a peculiar step ladder alignment of the trabeculae at the interdental septa. There are no alteration in the lamina dura or periodontal membrane (345). Similar findings were reported by Morris and Stahl (301), Mittleman et al and by Prowler and Smith (328), not only in patients with sickle-cell anemia, but also in many with only the sickling trait.

Patients with cyclic neutropenia may exhibit cyclic, severe gingivitis which eventually may result in mild to severe loss of superficial alveolar bone even in children (66), Necrotizing ulceration of gingivae is sometimes observed in patients with agranulocytosis; the absence of a notable inflammatory reaction because of lack of granulocytes is a striking feature (269).

Gingival and periodontal manifestations occur with great frequency in acute and subacute monocytic leukemia, less frequently in acute and subacute myeloid or lymphoid leukemia and seldom in chronic leukemia (42,92,138). In acute form, the most common periodontal manifestations are marked gingival hyperplasia, gingival swelling due to infiltration of leukemic cells into marginal and attached gingiva, gingival hemorrhage resulting from ulceration of the gingival sulcus epithelium and necrosis of underlying tissue, and rapid loosing of the teeth due to necrosis of the periodontal membrane (42,92). In chronic leukemia, generalized alveolar bone resorption, absence of the lamina dura and diffuse and irregular periodontal space may occur (27). Difference in the degree of local irritation accounted for the great variation in the periodontal manifestations seen in different patients (138).

One of the prominent manifestation of thrombocytopenic purpura is severe and often profuse gingival hemorrhage which occurs in majority of cases. The gingiva is swollen, soft and friable, which again varies in severity by the degree of local irritation (138).

Hence, sickle-cell anemia, cyclic neutropenia, agranulocytosis, leukemia and thrombocytopenia can progressive periodontal disease. More studies, however, are needed to assess the extend to which local prophylaxis can prevent or delay such progression.

I.3.7.2.7 Other systemic conditions and disease

By comparing the severity of periodontal disease in hospitalized patients with different systemic diseases, several investigators have reported that patients with malignant neoplasm and liver cirrhosis had unexpectedly high periodontal scores (6,371,373).

Persons with gastrointestinal disorders also tended to have more severe periodontal disease (6,373). Results from a population survey in Tecumseh, Michigan indicated an inverse association between FEV1. and the severity of periodontal disease (430,431). Gould and Picton found that cyanotic children and young adults had more gingival abnormalities and deeper gingival pockets than their controls who had a similar age distribution, but the cyanotic subjects in general also had more dental deposits than did the controls (150). Groen et al observed that of 38 presenile osteoporotic patients whose ages ranged from 43 to 78 years, only two subjects had no sign of periodontal disease, 27 subjects presented a terminal stage of periodontal disease and the remaining 9 subjects had all teeth in the mobile state. Although the authors did not include control subjects for comparisons, the remarkable severe periodontal disease among those osteoporotic patients was impressive (158). In their study of periodontal disease among 381 employees of the Department of Hiways in Ontario, Freedman and his co-workers failed to find a significant association between allergic history and periodontal scores. Age or oral cleansing was not considered in the comparisons.

Although studies on diabetic persons (218) and mongols (394) revealed no particular relation between blood citrate levels and the degree of alveolar bone loss, higher blood citrate levels were reported to be associated with greater loss of alveolar bone in study on 30 females with periodontal disease (413). Subjects who had severe early destructive periodontal disease

(445) and periodontosis (446) had higher blood citrate levels than their age-matched controls. It was, however, not certain whether or how blood citrate acted in the pathogenesis of periodontal disease. There was no significant association between serum cholesterol levels and periodontal scores following standardization for age (211,430,431). Kelly and Engel reported that a significant rise in periodontal scores among men was associated with traces of albumin in the urine (211), It should be noted, however, that the actual number of people with albumin in the urine was quite small.

To conclude, subjects with malignant neoplasms, liver cirrhosis, obstructive pulmonary disease, albuminuria or presenile osteoporosis are likely to have more severe destructive periodontal disease than subjects without these or previously mentioned organic diseases. Evidence of association between the severity of periodontal disease and allergic diseases, gastrointestinal disease, genitourinary, or other respiratory disease are either weak or lacking (116,371,373).

I.3.7.2.8 Summary of organic systemic diseases

From the present review of the interrelation between periodontal and systemic disease, it seems certain that periodontal disease tends to become more severe in persons with the following disorders or diseases: poorly controlled diabetes, mongolism, other mental defects or retardation, cerebral palsy, increase of systolic blood pressure, peripheral arterial disease, retinal vascular changes, sickle-cell anemia, cyclic neutropenia, agranulocytosis, thrombocytopenia, leukemia, malignant neoplasms, liver cirrhosis, obstructive pulmonary disease, albuminuria and presenile osteoporosis.

The associations between periodontal disease and the following disorders or diseases are less convincing: mild or well- controlled diabetics, glucose tolerance within normal range, coronary or hypertensive heart disease, elevated diastolic blood pressure, arteriosclerosis, allergic diseases, gastrointestinal diseases, genitourinary diseases or other respiratory diseases.

This summary is by no means exhaustive. Knowledge of the interrelation of periodontal and systemic diseases is still far from complete. This is mainly the result of a failure in many previous investigations to study systemic disease on the basis of a disease entity or of a specific pathologic process involved. Too often our studies of periodontal and systemic disease have made comparisons involving such ill-defined categories of systemic disease as cardiovascular disease, gastrointestinal disease, respiratory disease, kidney (373) or endocrine disease (371). Another reason for our lack of knowledge of interrelation of periodontal and systemic disease is the incomplete history record about chronic systemic disease as well as chronic periodontal disease, which make the interpretation of the relation between the two diseases very difficult.

I.3.7.3 Mental illness, psychological factors and personality

The studies about mental status and periodontal disease can be discussed according to three types of populations.

I.3.7.3.1 Studies on emotionally disturbed subjects

Miller et al administered the Minnesota Multiphasic Personality Inventory (MMPI) to 50 clinical patients and found a postive, though not significant, correlation between periodontal disease and anxiety (293). However, their study lacked proper controls. Baker and others(16) conducted a study on 26 psychotics,36 psychoneurotics and 40 normal subjects to investigate the relationship between personality factors assessed with the MMPI and the severity of periodontal disease measured as alveolar bone resorption and pocket depth. The results indicated a positive relationship between periodontal disease and factors such as age, broken home, marital adjustment, somatization, hysteria and the MMPI scale. The authors (16) concluded that difficulty in personality adjustment might correlated with periodontal disease. Studies by Belting and Gupta (23,24) on 104 psychiatric patients and 122 patients for medical care other than psychiatric treatment revealed that the former had greater severity of periodontal disease in all age groups, and such differences remained even after brushing frequency, amount of calculus and the habits of bruxism and clenching were held constant in the two groups. When only psychiatric patients were considered, the severity of periodontal disease increased significantly as the degree of anxiety increased. Davis and Jenkins (81) tried to correlate emotional stress, as measured with the MMPI, and PI scores among 89 patients who were admitted to a psychiatric center. A simple correlation co-efficient showed that there were significant associations between PI scores and emotional factors, ie; depression, schizophrenia, mania and Welsh anxiety. By means of partial correlation co-efficients, they further demonstrated that anxiety was the main element in schizophrenia, depression and mania which led to the high PI scores. The authors also reported that factors of "oral hygiene" (not defined) and age could not explain the relationship between degree of anxiety and PI scores.

I.3.7.3.2 Studies on subjects with periodontal disease

Manhold and Manhold (268) studied 50 subjects with periodontal disease in the Great Lake Naval Training Center. They observed that the persons whose neurotic or introversion-extraversion tendency percentiles were above the third quintile, as compared, as compared with those below it; presented more severe periodontal disease. Since the psychological tests employed in this study were considered to be of doubtful validity, and the arbitrary dichotomy of severity of periodontal disease invited great observer error, the validity of this study remains a question. A detailed case history study was carried out by Moulton et al on 6 cases of acute necrotizing gingivitis and 16 cases of chronic periodontitis (303). Acute necrotizing gingivitis was, in all instances, precipitated by acute anxiety arising from a life situation of conflict about dependency and/or sexual needs. Local factors and oral habits were minimal. The subjects with chronic periodontitis had a background of longstanding, less acute conflict, mainly involving dependency needs. Bleeding gum was noticed to occur at times greatest anxiety. Although their study was comprehensive, the small sample size and the lack of other quantitative data limited any generalization. By comparing personality states between subjects with acute necrotizing gingivitis and those with "other" gingivitis in 66 military subjects who had periodontal disease and were considered to be in acute emotional stress, Goldberg and his coworkers (147) reported no effect due to personality differences. However, on the basis of interviews, patients with acute necrotizing gingivitis tended to have more personality adjustment problems than those with other gingivitis. Barry and Dukovic made 4 psychological tests on 50 periodontal patients and compared the results with data from mormative groups (19). These tests were anxiety, neurotism, extraversion and authoritarian. No significant difference in any of the four psychological scores was found between their patients and the normals. Since the "normal" groups differed greatly from their patients in age, occupation and marital status, justification for such comparison was doubtful. The authors also doubted the severity of these four psychological tests. Recently, Formicola and others (114) made 15 personality trait tests on 41 subjects with acute necrotizing ulcerative gingivitis and other 41 subjects with healthy gingivae. All of them were preflight students in the indoctrination phase of training. The authors found that there was a significant positive correlation between scores of dominance trait and gingival scores and significant inverse correlation abasement trait and gingival scores. But the variance of gingival scores explained by these two variables was rather small, ie; 6.3 and 6.8 percent respectively.

I.3.7.3.3 Population surveys

Shiller reported from his study on 298 enlisted propective submariners, 19 to 32 years in age, that subjects who had high anxiety and/or low motivation brushed their teeth significantly less frequently and had more debris on their teeth than other subjects. But he did not find significant association between PI scores and either of these two emotional variables (408). This was possibly due to the fact the study subjects were relatively young and healthy and that the PI was not sensitive enough for measuring minor gingival changes which were commonly associated with emotional stress and oral cleansing. As part of their study to correlate social variables and periodontal disease among Negro high school students, Mobley and Smith (297) demonstrated a significant relationship between periodontal scores and schizophrenia on 72 subjects. Other personality traits which were also related with periodontal scores, though not significantly, were hypomania, psychasthenia, social introversion and depression. Their findings agree well to those of Davis and Jenkins (81). From their study on the U.S. adult population, Kelly and Engel (211) found a non-significant elevation in peridontal scores for both sexes who answered yes to having history of nervous breakdown. They also reported that for women, but not for men, there was a trend toward higher periodontal scores with increasing numbers of nervous symptoms.

Thus, a general impression from these studies is that when the study population consisted of mentally ill and normal subjects, some degree of association between periodontal disease and emotional stress could be found. If the study subjects were from periodontal patients or population sample, one had less chance to demonstrate such association. It, therefore, implies that emotional factors may contribute to severity of periodontal disease is relatively small. The mechanisms by which mental factors act on periodontal tissue were suggested and discussed without substantive evidence in two articles (160,343).

I.3.7.4 Puberty, Menstruation, Pregnancy and Menopause

I.3.7.4.1 Puberty

Puberty is often accompanied by increased gingival inflammation. Following puberty, the severity of the gingival reaction diminishes even if the local irritants have not been removed (138,422). From the fact that gingivitis is not a universal occurrance during puberty, it seems that physiological changes associated with puberty have increased the severity of gingivitis rather than initiating gingivitis. Pronounced inflammation and sometimes severe enlargement of the gingiva result from a degree of local irritation that would ordinarily elicit a comparatively mild gingival response, suggesting an exaggerated response to a local irritants at puberty (12,68, 228).

In a series of world-wide studies which included populations with age ranging from 5 to 55 years and over, there was a biphasic distribution of mean PI scores along age scale

(154,198,368). In general, before age of 20 to 25 years, mean PI showed little changes with age; after 25 years of age, PI scores increased precipitously. OHI scores showed a similar pattern of changes, which was attributed primarily to changes CI scores (154). In the case of CI scores, however, the slope was steepest in the pre- and early "teen" ages and its rate of accent decreased in the latter years. Furthermore, whereas the change of slope for PI scores occurred at approximately 25 years of age, such change for CI scores took place at an earlier age, ie; about 17 to 18 years. At present, no one can explain the biphasic distribution of mean PI scores besides its possible relation with calculus formation (421). A study by Vogel and Amdur suggested that a decrease in the concentration of inorganic pyrophosphate in the parotid duct saliva after puberty might be responsible for an increase in calculus formation (450).

Thus, puberty is frequently accompanied by an exaggerated response of the gingiva to local irritation and by an increase in calculus formation. Chronic destructive periodontal disease progresses rapidly after puberty.

I. 3.7.4.2 Menstruation

There is lack of good, controlled studies on the gingival changes during each menstrual cycle. Stoloff observed on oral syndrome (periodic transitory meno gingivitis) which occurred just prior to menstruation, in amenorrheas of different types, in post-hystectomy, prior to and after unruptured ectopic pregnancy, and during and after menopause. Such an oral syndrome consisted of discomfort, sensitiveness, redness and congestion, with bleeding under the normal stresses of mastication (428). Hirschfeld also reported recurrent gingivitis (menstruation gingivitis) appearing several days before the onset of the menstruation period, which is characterized by periodic recurrent hemorrhage with bright red and rose-coloured proliferations of the interdental papillae (170). Lately, Lindhe and Attstrom observed a definite increase in gingival exudate at ovulation (244). Prout and Hopps studied six healthy female students aged 20 to 22 years, whose samples of saliva were collected daily over two and a half months and were counted for total bacteria. They found an increase in salivary bacterial counts during menstruation and at ovulation (327).

Sheiham found no marked difference in PI scores between 289 pre and post-menarche girls aged 11 to 14 years, although there was a slight tendency for PI scores to be lower in girls who commenced menstruation (49,339) or 25 months before than non-menstruating girls of same ages (402).

Since PI is not a sensitive index for minor gingival changes, it is not known whether significant differences in the severity of gingivitis between two groups of girls can be demonstrated if the gingivitis index had been used instead. From the three preceeding studies (170,244,327), there is periodical fluctuation of some oral parameters as a function of menstrual cycle. The time for gingival assessment on each girl after commencement of menstruation, in Sheiham's study (402), may also affect the gingivitis score and the comparison with non-menstruating girls.

Thus, there seems to be a periodic fluctuation of salivary bacterial counts, a variation of gingival exuadate and changes in the nature of gingivitis as a function of the menstruation cycle. There is, however, a lack of quantitative data about the severity of gingivitis which demonstrate any definitive fluctuation in gingival disease in relation to the menstruation cycle.

I.3.7.4.3 Pregnancy

Maier and Orban studied on 530 pregnant women aged 16 to 42 years and found that the severity distribution of gingivitis did not differ significantly from that of non-pregnant subjects. Clinical and histopathologic features revealed no diagnostic or characteristic findings in pregnant gingivae (264).

There were eight other cross-sectional studies of periodontal parameters among pregnant and postpartum or control subjects (98,207,248,251,338,344,410,411). A study by Ringsdorf et al revealed significantly greater tooth mobility among pregnant than postpartum women (338). But there was no significant difference in PMA gingivitis counts either between pregnant and postpartum subjects or among pregnant women in the third trimesters (344). Loe and Silness (248,251,410) studied 121 pregnant and 61 postpartum women and found that the pregnant women had higher GI,PI and deeper pockets. GI and PI scores increased at the second month of gestation, reached a second peak at the eighth month and decreased during the last month of pregnancy. No such marked fluctuation in pocket depth was found. The quantity and nature of oral debris did not differ between pregnant and postpartum women. It was interesting, also, to find that correlation co-efficients of GI scores with PL I(Plaque Index) scores and/or with CI scores were much lower during pregnancy than postpartum, suggesting that during pregnancy some factors, in addition to amounts of dental plaque and calculus, contributed to the greater severity of gingivitis. Changes of gonadotropin, estrogen, progesterone and relaxine during pregnancy were speculated by the authors to be some of the responsible factors (248). Results from the same study (251) also suggested that the increase in pocket depth at pregnancy was mainly due to gingival enlargement rather than apical movement of the epithelial attachment, because of the fact that pocket depth decreased significantly after parturition and that there was no significant difference between pregnant and postpartum groups with regard to occurrence of destructive periodontal disease. A further study by the same authors (411) on 33 pregnant women who received conservative local therapy, consisting in removal of hard dental deposits and correction of overhanging margin of dental restoration, revealed that gingivitis could be abolished and normality maintained during pregnancy provided the gingival areas of the teeth were kept clean. Such finding also suggest that although bacterial plaque was responsible for the initiation and maintenance of gingival inflammation in pregnant women, the altered tissue metabolism at pregnancy accentuated the inflammatory response. It is, however, wise to keep in mind that these were cross-sectional studies, and that for each month of pregnancy and postpartum, there was only 10 to 20 subjects whose age distribution was not stated. How much would this variable, age distribution, could contributed the variation in gingivitis scores at different stages of gestation and postpartum remains unanswered.

Two other cross-sectional studies (98,207) were designed to compare the periodontal parameters between pregnant and non-pregnant women of comparable age, as ~~well~~ as in pregnant women during their three trimesters. Both studies revealed significantly higher gingivitis scores among pregnant than in the control women. While one study (98) showed gingivitis scores of the women in their third trimester to be remarkably higher than those in the first and second trimesters, no consistent trend of gingival scores of the third to nineth month of pregnancy could be found in another study (207).

Cohen and others (64) carried out a longitudinal study on 16 pregnant women. Each study subject was examined at the end of three trimesters and three months postpartum. Results showed that the gingivitis scores increased throughout the pregnancy and partly decreased three month postpartum. Only negligible changes in epithelial detachment could be noted. Hard deposits also increased throughout the pregnant and postpartum, but there was no consistent trend in the build-up of soft debris. Their findings of lower correlation co-efficients between soft debris and gingivitis scores as well as between hard deposits and epithelial detachment at pregnancy in comparison with the same situation in postpartum women agreed well with the studies by Loe and Silness (248,410). But Cohen <u>et al</u> (64) also reported that correlation between hard deposits and gingivitis score was higher at pregnancy than at postpartum, which was contrary to the findings by Loe and Silness (248,410). In a further study by Cohen et al, each pregnant women was age-matched to a non-pregnant control, and an additional examination was made in fifteenth month postpartum. Analysis of variance revealed that both pregnant women and their controls had significant changes during the five examinations in Gingival-Periodontal Index (GPI) and CI, but no significant changes in epithelial detachment and Plaque Index (PL I). While pregnant women had significant changes in tooth mobility but not in GI during the five examinations, their controls demonstrated changes of these two parameters in the opposite direction. When the means of each parameter between pregnant women and controls were compared for each examination, significantly higher GPI,GI and tooth mobility among pregnant women at various stage were found. Such differences were not found for CI,PLI or epithelial detachment. These findings, therefore, suggested that the main periodontal pathosis associated with pregnancy was gingival inflammation rather than epithelial detachment, that increase in soft or hard deposits and that an increased severity of gingivitis at pregnancy was maintained fifteen month post-partum. These findings again agreed very well to those reported by Loe and Silness (248,251,410). It should be understood, however, that none of oral assessments of pregnant women were made blindly. Slome studied periodontal disease on post-partum Jewish women in Israel(415). For the age group 25 to 29 years, periodontal score was found to be higher among subjects with parity of 6 or over than those of 1 to five. It was not clear whether this difference came from incomplete recovery of periodontal disease between pregnancies among women with more children, or from other socioeconomic differences between women with large and small families.

In conclusion, pregnancy is associated with greater severity of gingivitis which can not be explained by corresponding increases in oral debris or calculus deposition. Increased severity of gingivitis seems to have no lasting deteriorating effect after delivery. There is no consistent finding with respect to the fluctuation in severity of gingivitis throughout pregnancy; monthly assessment on a cohort group is needed to clarify this controversy. Gingivitis can be abolished and normality maintained during pregnancy provided that gingival areas on the teeth are dept clean --- suggesting that pregnant state is a modifying rather than an initiating factor in gingivitis. Pregnancy is not associated with apical migration of the epithelial attachment.

I.3.7.4.4 Menopause

There is a lack of research concerning the effect of menopause on periodontal disease. Data from population surveys did not allow for any estimate of the menopausal effect on periodontal disease due to crude age groupings such as 50 or 55 years and over, for example, and the few study subjects in the older age groups (154,198,368). Data from population surveys on U.S.

adults, however, indicated a steeper increase of PI score in women from the age group of 35 to 44 years to those of 45 to 54 years than those before 35 years old. No similar change tooth place in men of comparable ages (212). Recently, a cross-sectional study was made by Hochman et al among 30 pre- and 30 postmenopausal women (171). They found that the formers had, on the average, significantly lower OHI and PI scores and fewer teeth lost. Significant correlation co-efficients between OHI and age were also found. The authors did not employ analysis of covariance to compare, between the two groups, PI or OHI adjusted for age which very likely differed between the two groups. They did, however, found that 9 older women in the reproductive cycle as compared with 10 women shortly after menopause had a significantly lower PI score but not OHI score, suggesting menopausal effects on PI score without corresponding change in the OHI score. One must realize that it was not a cohort study and that the data were based on 10 or fewer study subjects; generalization from this study, therefore, is very limited.

Hence, from very limited data, there is an indication of greater severity of periodontal disease among postmenopausal women than one might expect. More investigations with better designs are, however, needed before the menopausal effect on periodontal disease can be more precisely stated.

I.3.8. Oral hygiene factors

I.3.8.1 Studies on severity of periodontal disease in relation

to frequency and technique of oral cleansing. McHugh and his associates reported that the average OHI scores of the 13 years old Dundee school children decreased significantly as toothbrushing frequency increased (282). Studying the interrelation of toothbrushing frequency and plasma level of ascorbic acid with respect to the dental debris score in adult subjects, Clark <u>et al</u> found that among subjects who had optimal levels of plasma ascorbic acid, toothbrushing frequency was signigicantly and inversely associated with debris score. No significant association was found among persons who had rather low levels of plasma ascorbic acid (63).

Studies by Greene (153) and Ramfjord (330) on Indian children aged 11 to 17 years revealed that most Indian children cleansed their teeth with fingers and abrasives, and only a few used a toothbrush or stick. Although the data showed that toothbrush users tended to score lower on the PI than those used either fingers or stick, no statistically significant difference in average PI scores among the three groups was found, due possibly to the small number of toothbrush and stick cleansers. Held compared the amount of oral deposits and the severity of periodontal disease between those who cleansed their teeth with fingers and those employed a toothbrush in Iranian teenagers and adults. He found a significantly higher PI and OHI scores among finger cleansers than toothbrush users for comparable age groups (164). As part of a clinical trial to compare the effect of toothbrushing frequency on gingival health, Swallow and his associates observed no significant difference in average gingivitis scores between subjects using manual as against automatic toothbrushes at the end of three experimental weeks (438).

Greene's study on Indian children also showed no significant difference in average PI scores between children who cleansed teeth with varying frequency.(153). Such results might be due to varying cleansing methods as well as their unpredictable efficiency. Belting and Gupta observed that among psychiatric patients or patients with other physical illness, the average PI score was higher among subjects who brushed teeth less frequently. Such a trend was not statistically significant which might be due to not controlling the age factor which ranged, in their study subjects, from 20 to 65 years (24). Similar findings were also reported by Belting et al in their study on diabetics and patients with other physical illness (25).

Studies conducted upon young adults in Singapore (279), pregnant women in Alabama (344), postpartum Jewish women in Israel (415). enlisted prospective submariners in U.S.A., (408), and Melbourne residents in Australia (243) have demonstrated a significant inverse association between average periodontal score and toothbrushing frequency.

Toothbrushing frequency has also been correlated with both periodontal score and amount of oral deposits simultaneously in cross-sectional studies upon Norwegian army recruits (38), young soldier in Sydney, Australia (79), white school children in Atlanta, Georgia (414), Adventist and non-Adventist teenagers in Tennessee (172), some Caucasian adult patients from three clinics in U.S.A. (176), Surrey school children in Kingston, England (402), and institutionalized inmate adults(432). The unanimous significant inverse correlation of toothbrushing frequency with scores of periodontal disease and oral debris in all these studies was truly convincing.

The association between toothbrushing frequency and calculus score, on the other hand, was less predictable from one study to another (38,172,402). Since rate of calculus formation, especially supragingival calculus, differs from one person to another irrespective of oral cleansing (450), and calculus can hardly be removed from the tooth surface through toothbrushing once it matures, the inconsistent association between calculus and toothbrushing frequency is not unexpected.

Withdrawal of oral hygiene measures always resulted in an increase in prevalence (173) and severity (117,252,426) of gingivitis and in the dental plague (117,252) and calculus score (426). When oral hygiene measures were readministered without long delay following the appearance of gingivitis, all soft deposit as well as most of the newly formed loose calculus could be easily removed with a toothbrush and the deteriorated gingival tissue could recover to the initial state (252,426).

Giving instruction of toothbrushing methods to various populations has been shown to improve gingival and periodontal health when compared with non-instructed subjects (38,39,226, 317). A corresponding reduction in the amount (38,39) or change in quality (226) of oral soft deposit among instructed subjects was also observed. Swallow <u>et al</u> conducted a study on 80 mentally handicapped adults in which every group of 20 subjects received toothbrushing care by two operators either once daily, twice weekly, once weekly or not at all (as a control group). At the end of the three week-experiment, the authors found that whereas the control group did not change much in GI score, all experimental subjects had improved their gingival health. There was no statistical difference in the reduction of the GI scores among the three experimental groups, due possibly to the great differences in their initial GI scores (438). Goyings and Riekse brushed the teeth of their experimental subjects for a total of 60 days following an initial oral prophylaxis which was given to both experimental and control groups. They found that only experimental subjects had significantly improved periodontal health and reduced amounts of plague and calculus (151). Lovdal <u>et al</u> reported that periodontal scaling and toothbrushing instruction given to industrial employees for fove years had markedly reduced the prevalence of gingivitis. The authors, however, added that the absence of pathological pockets was essential for success (254). Silness and Loe demonstrated that initial prophylaxis, correction of overhanging margins of dental restoration and toothbrushing instruction had consistently reduced mean scores of plaque, calculus, GI and pocket depth among women in early pregnancy (411). Lighter <u>et al</u> made a clinical trial on young men in U.S.A.F. and observed that the plaque score was reduced mainly through toothbrushing instruction, hard deposit through more frequent scaling and that both the gingivitis score and the Irritant Index score (materia alba, calculus and/or subgingival overhangs of dental restoration) were reduced through more frequent scaling and toothbrushing instruction. The authors, however, reported no change in PI score throughout a 46 month-experimental period (239,241), possibly because of relatively young age of the study subjects and the scarcity of subjects with destructive periodontal disease, thus making the index not really sensitive to the improvement in periodontal health of this study population. Although the same authors also reported that subjects who had received toothbrushing instruction and more frequent scaling annually, had a lower incidence of epithelial detachment at the end of experiment, it had little meaning because no initial data about this parameter was available (241). Suomi *et al* conducted a similar trial on industrial employees in which the experimental subjects received annual toothbrushing instruction as well as five scalings in the first year, four in the second year and three in the third year. The control group did not receive toothbrushing instruction or scaling. Both groups were initially matched in pairs according to periodontal score, oral hygiene status, caries, age and sex giving a predetermined weight for each factor. Results showed that while the experimental group did not change much in their scores of debris, suprasubgingival calculus, gingivitis index, or in degree of epithelial detachment and alveolar bone loss; the control group had consistently higher in average scores for all these indices and parameters (433,434,436).

Stallard and his co-workers put dental students on one of three oral hygiene programs, i.e., toothbrushing, control mouth rinse or antibiotic mouth rinse, at a weekly shifting schedule for a total of seven weeks. They reported that because of relatively low initial scores of plaque, calculus and gingivitis index, toothbrushing did not greatly improve these scores; but control mouth rinses without toothbrushing always resulted in an increase in all these indices, and antibiotic mouth rinses allowed plagued accumulation but maintained the same amount of calculus and same degree of gingivitis (426). In a study to find relative effects of oral prophylaxis and protein supplementation on gingival health, Cheraskin et al reported that whereas protein supplement with or without oral prophylaxis significantly reduced gingivitis scores, oral prophylaxis alone did not significantly improve gingival health (60). Since the protein supplemented group had initially a higher average gingivitis score than the placebo group and the final average gingivitis score of two groups were practically the same, it is difficult to interpret the effect of protein supplement on gingivitis if fluctuations of gingivitis which can occur during a two week period (174,435) were taken into account. However, it was obvious that oral prophylaxis alone without toothbrushing instruction did not significantly promote gingival health.

To summerize, if oral cleansing methods are relatively standardized, more frequent oral cleansing is associated with a decrease in oral soft debris, better gingival health and lower score in PI. The relationship between frequency of oral cleansing and the amount of calculus deposition is much less consistent. There is no sufficient evidence that oral cleansing frequency is associated with alveolar bone loss or epithelial detachment. Withdrawal of oral cleansing always resulted in more accumultation of plaque and calculus and a greater degree of gingivitis. Readministration of oral cleansing without long delay can restore the tissue back to their original condition. Absence of true periodontal pockets, correct oral cleansing methods, more frequent cleansing, and periodical oral prophylaxis (scaling) by dental professionals, are all essential for removing both soft and hard oral deposits and for deeping healthy gingivae. A mouth rinse with water alone is not sufficient for oral cleansing, but antibiotic mouth rinses may help to maintain gingival health. The advantage of the automatic over the manual toothbrush in oral cleansing is not substantiated.

I.3.8.2 Studies on severity of periodontal disease in relation to oral deposition

Studying on industrial workers and staffs, Arno, Lovdal, Schei and their co-workers found that, for each age group, the amount of soft deposit on tooth surface was significantly associated with the prevalence of gingivitis (9,255), pocket depth (255), percentage of mobile teeth and of alveolar bone height (256,375). Same authors also reported a positive correlation between subgingival calculus and gingivitis scores (255). Mehta et al observed from Ontario civil servants in Toronto, that average number of pocket per person and per tooth increased with age, and that efficiency of oral hygiene measures, according to amount of oral deposits, became worse as age increased (289). These facts certainly did not, in themselves, proved an association existing between amount of oral deposits and prevalence of pocket formation. Same authors, however, also found that the proportion of subjects having more oral deposits were greater among those who had complex periodontitis which was a more severeform of periodontal disease (289).

O'Leary et al found that among newly recruited young subjects the mean plaque score was greater in those who had no gingival recession than those had. It was thus postulated that more vigorous and thorough toothbrushing among this study population had resulted in less plaque, healthier gingiva and presence of gingival recession (309).

Following the introduction of the Oral Hygiene Index (OHI) which measures the amount of oral debris (DI) and calculus (CI), by Greene and Vermillion (156) and later of the Simplified Oral Hygiene Index (OHI-S) by the same authors (395), many studies have shown that average scores of PI, CI and OHI increased with age (1,212,213,395,396,469), which can only be taken as a suggestion of an association between scores of PI with both OHI and CI. When a population of smaller age range was studied, there was no consistent pattern between age and either PI, CI or OHI (402).

The mean severity score of periodontal disease had been observed to increase in the same direction with mean score of OHI and its intermediate indices, i.e., DI and CI (24,25,38,153, 201,213,221,243,247,261,279,280,282,332,361,366,395,402,408,415, 444,463). Age factor was not standardized in some of these studies despite the fact that it varied greatly among study subjects, the relation between severity of periodontal disease and OHI score would therefore be subjected to various interpretations (24,25,332,415). All other studies have considered age factor by studying population of small age range (38,153,201, 221,247,279,280,282,408,444), or by comparing the severity of periodontal disease among persons at various OHI score intervals but belonged to same age category (213,366,395,396,402, 463), or by comparing the percentage of alveolar bone loss adjacent to tooth surface with calculus than that without calculus (261). Of the reports which have shown correlation of both scores of CI and DI with PI score, average CI scores showed better discrimination, than DI score, among different severities of periodontal disease within each study population (53, 213,221). Study made by Lilienthal et al in Melbourne, Australia revealed that for males and females aged 5 to 9 years, oral debris alone influenced the PI score, that up to age of 20 years for females and 24 years for males, both oral debris and calculus strongly influenced the PI score and that in older age-group only calculus affected the PI score (243). The OHI score has also been shown to explain different severity of periodontal disease associated with sex, race, income, education and occupation (213,361,366,463). Waerhaug observed that although Norwegian recruits, aged 19 to 21 years, as compared with Ceylonese male students of the same age, scored much lower in average PI score which were 0.80 and 1.14 respectively, when these populations were distributed according to OHI scores, the PI scores of the two populations within equivalent OHI groups were very much the same (463). This interesting phenomenum was confirmed later by Johansen in his study on dental students both in India and Oslo whose mean PI scores were 1.184 and 0.319 respectively, though in this case PI scores of the two populations with equivalent DI scores, instead of OHI scores were compared due to the fact that Oslo students had practically no dental calculus (186).

The simple correlation co-efficient of PI and OHI scores has been estimated in many studies (38,93,109,154,156,176,201, 368,388,395,396,409,444). The values ranged from 0.50 (444) to as high as 0.85 (395), depending, partly, on the age ranges and specific social characters of the study populations. In general, the greater the age range of a study population, the better the correlation between PI and OHI scores existed (149,368,396). More privileged population when compared with less privileged ones, tended to have greater correlation between scores of PI and OHI too (109). When only young subjects were studied, PI scores tended to correlate better with DI score than with CI score (38,154,156,414). But when population of whole age-ranges or especially only older subjects were considered, PI scores correlated much better with CI score than with DI score (109,154, 176,201,414). Loe's plaque Index score has been found to be highly correlated with his own Gingival Index score (410) and Periodontal Score (409). The correlation between Plaque Index and Gingival Index was much greater than that of Calculus Index and Gingival Index among women at pregnancy and postpartum (410). The OHI score has also been reported to correlate positively with Gingival Recession Score (18,368) as well as degree of alveolar bone loss (394).

Since there are enough evidences that factors such as age, OHI and other speculated etiological variables are not in themselves independent one from another (18,109,154,247,368,394,409), technique of multiple correlation co-efficient has been employed to estimate the proportion of severity variance in periodontal disease which can be explained by combined effects of all these variates (18,109,154,247,368,469). Studies made in Chile (18), Ethiopia (247) and South Vietnam (368) had taken factors of age, sex, OHI score and five or more biochemical data of nutritional status into consideration. In general, about 50% of the variance in PI as well as Gingival Recession Score could be explained by combined effects of these variates, and factors of age and OHI score alone accounted for about 90% of the variance explained (18,247,368). There was a definitive phenomenum that variance in Gingival Recession Score was more accounted for by age than by OHI, whereas PI variance was either equally explained by age and OHI (18) or more explained by OHI than by age factor (247). In his study in South Vietnam and Lebanon, Russell concluded: "Less than 10% of the variance in group PI scores remain to be explained after the combined influence of age and mouth cleanliness has been estimated."(368). Since this author estimated the multiple correlation co-efficient from "group scores" of PI and OHI about which he did not specify, rather than scores of PI and OHI of individual subject, hence, his statement: " a residual factor wholly independent of age and oral hygiene, therefore, can have little effect on periodontal disease as scored by PI," (368) is really not justified. Other studies also revealed that the proportion of the variance in PI scores which was accounted for by factors of age, DI and CI ranged from 30% among Osegere Villagers in Nigeria (109), 55% among 456 South

Vietnamese (368), 58% among Ecuadorian civilians and Montana Indians (154) to as high as 81% among Yoruba family members in Nigeria (109).

Thus, prevalence, especially severity, of periodontal disease is associated with the amount of oral debris and calculus. Such correlation is stronger if study population vary greatly in age, are more privileged in material needs and belong to higher social status. Oral debris seems to be more associated with gingivitis at younger age, whereas calculus emerged as stronger or even the only associated oral deposit with more severe form of periodontal disease as population become older.

I.3.8.3 The fallacies of using the Oral Hygiene Index and other similar indices as a measure of oral cleansing effect

Following the introduction of the Oral Hygiene Index (OHI) (156) and Simplified OHI (157) by Greene and Vermillion, the term " oral hygiene" has been loosely used to denote both oral cleanliness as well as oral cleansing. The following quatations examplify this quandary: "Prophylaxis and improvement of oral hygiene may decrease the average mobility as much as 25 percent" (462). "A residual factor wholly independent of age and oral hygiene, therefore, can have little effect on periodontal disease as scored by PI." (368). The OHI is introduced as a measure of the amounts of soft debris (DI) and calculus (CI) on the tooth surfaces (156), it, therefore, is not synonymous with oral cleansing effect.

Despite the fact that studies both from case history and clinical trial indicate less amount of oral debris deposition can be achieved from more frequent tooth cleansing (38,39,79,117, 156,172,176,239,259,402,411,432,433,434), amount of calculus is however less consistently or not at all related to frequency of tooth cleansing.(38,39,156,259,402,432). Even debris formation is partly depended on nutrition (58,63,69,75,96). Although it is now well accepted that the soft, adherent, non-calcified plaque containing myriads of micro-organisms represents the initial phase of supragingival calculus formation which is subsequently and rapidly calcified (266,448), supragingival calculus has also been found to be related to salivary viscosity (371), inorganic pyrophosphate concentration in the parotid saliva (450), vitamin A deficiency (368) as well as coronary heart disease proneness (471). Study made by Lightner et al on U.S.A.F. new recruits revealed that while toothbrushing reduced plaque accumulation, calculus was removed mainly through frequent scaling (239). Suomi et al also conducted a clinical trial on industrial employees in which the experimental subjects received three or four scalings and toothbrushing instruction annually, the authors found that while debris score of the experimental years, their supra- and sub-gingival calculus increased rapidly during the first two years.(433,434). Such descrepancy between oral cleansing frequency and amount of dental deposits, especially dental calculus was also observed among rural Maori population who hardly brushed their teeth and yet had relatively little calculus in contrast to great amount of debris (243), among adventist teenagers who, as compared with non-adventist teenagers, brushed more oftenly and yet had more debris and calculus (172), and among Atlanta teenagers who scored higher in DI but much lower in CI than urban Indian teenagers (153).

We have very little knowledge about the exact role of the subgingival calculus in the pathogenesis of periodontal disease. We are still far from reaching an agreement whether subgingival calculus if the cause, the result or both in the disease process (7,169,265,422).

Hence, indices which score the amount of dental deposits actually measure not only oral cleansing effect, but also dietary, nutritional and constitutional factors. In addition, these indices may even measure part of the periodontal disease itself. Therefore, taking amount of dental deposits as an index of oral cleansing effect in the epidemiological studies of periodontal disease will result in misinterpretation of the pathogenesis of this disease and in less effectiveness from preventive measure subsequently.

I.3.8.4 The fallacies of evaluating the association of periodontal disease with variables other than the OHI score and age by standardization of the latter two variables.

As it is discussed at the preceding section that amount of dental debris and calculus are to some extent the functions of factors such as nutrition (18,58,63,69,75,96,247,368), diet (172), saliva (379,450) and other constitutional variables (471). Nutritional factors (18,247,368) and general metabolism (430) are also closely associated with age factor. It follows, then, that debris, calculus and age can not be held constant when evaluating those systemic factors as potential etiological factors of periodontal disease. Instead, one should study the effect of systemic factors on periodontal disease among people of similar age who practise at various degree of fairly standardized oral hygiene care --- an analogous study design of analysis of covariance.

In addition, if the formation of subgingival calculus is part of the result rather than the cause of the chronic destructive periodontal disease,(7,169,265,422), and if its amount measured from cross-sectional study also reflects partly the duration of the existing disease or an indirect measure of age of each study subject, it, then, seems very unlikely that following the standardization for subgingival calculus score and age, there is much variance in PI score remaining to be accounted for by other suspected etiological factors --- a mechanical phenomenon of step-wise regression analysis. And yet such finding dose not contribute to the knowledge regarding the real etiology of periodontal disease. Unfortunately, many data from cross-sectional studies have been analized in this fashion and concluded no evidence of association existing between periodontal disease and nutritional factors (18,247,368,463,469), or systemic metabolism (430).

Thus, there are great deal of data to suggest that an individual's OHI score is, to some extent, a function of nutritional, dietary and sustemic factors as well as an indirect measure of age, that subgingival calculus may be the result rather than the cause of the periodontal disease and that individual's nutritional condition also depends on age. Evaluating the association of periodontal disease with variables other than OHI score and age by standardizing the latter two variables, such as being conducted in most epidemiological studies of periodontal disease has, therefore, resulted in possible erroneous conclusion regarding the true relationship between periodontal disease and other suspected etiological factors.

I.3.9 Effect of Smoking, Betel nut chewing and Alcohol drinking

I.3.9.1 Effect of smoking

Reference (320) to a relationship between smoking and periodontal disease date back to 1859. However, relatively little research regarding this relationship had been reported in the literature until last 25 years. There is now a general consensus that smoking tended to increase the prevalence of ulceromembranous gingivitis (133,287,320), simple gingivitis (9,115,320,321), active periodontal disease as measured by PI or PDI (38,403,420, 430,431) as well as destructive phase of the periodontal disease (8,403), corresponding to this association is a similar relationship existing between cigarette consumption and amount of soft deposit (8,9) hard deposit (115,320,321) or both (38,403).

Other studies reported a similar trend regarding the associations of cigarette smoking with periodontal disease and oral deposits, but failed to demonstrate a statistical significance of such trends(164,201,408).

Some of the studies mentioned above have been tried to standardize the factors of age and/or oral deposits, thus, to estimate the residual association between cigarette consumption and periodontal disease. The results revealed either reduction, though still remaining to be significance of (8,9,321,430) or nearly absence of (38,243,403) the original correlation between cigarette consumption and periodontal condition. Two of these studies (8,38) also showed that the effect of smoking reduced to minimum in persons who had "good oral hygiene". Such phenomena are interpreted as that the effect of smoking on periodontal disease is not as great as "oral cleanliness" (266), without considering the possibility that both soft and hard deposits may be, partly, the result of smoking and even periodontal disease itself, and that a smoker may require more effort than non-smoker to keep an equivalent amount of oral deposits.

I.3.9.2 Effect of betel nut chewing

Betel is a leaf of a tropical tree. In this is packed the areca nut, some tobacco, various spices and lime. The package, or quid, is put into mucobuccal fold. It is consumed mostly by Eastern people. Mehta et al found that the teeth of the mandibular left side where the quid is kept were more affected by periodontitis and subsequent alveolar bone resorption than the teeth of the right mandibular side (287). Gupta's study in Trivandrum, India confirmed this finding in which he use PI as a measure of active periodontal disease (159).

In a survey, comprising about 8,000 Ceylonese, Waerhaug (463) observed that, for the subjects of 20 years old or above, betel consumers consistently scored higher average PI than non-consumers of the same age groups. Corresponding to this is the higher proportion of the betel chewers than non-chewers to have higher OHI-S scores. When the betel chewers and non-chewers of the like age and OHI-S scores were compared, the former remained to score higher PI than the latter in most comparisons, though their differences reduced to a lesser degree. It is also interesting to find, that this same study (463), the smokers consistently had lower PI scores than non-smokers. However, the author observed that the alternative to tobaccosmoking is very often betel chewing in this study population. Therefore, the group of tobacco smokers has in its control group all the betel chewers, and the control group with whom the betel consumers are compared, contains most of the tobacco smokers. Obviously, betel consumption is a more destructive factor than is tobacco smoking. Most recently, Johansen conducted a dental survey on some Indian dental students (201). There were only 11 male betel chewers who also smoked cigarette. They were compared with 25 male smokers who did not chew betel. The results indicated the former, as compared with latter, scored, in average, higher PI,CI-S, DI-S. None of these comparisons was significant statistically, possibly, due to small number and relative young age of the study subjects.

It seems fair to conclude that betel chewers than non-chewers tend to have higher prevalence and severity of periodontal disease as well as more amount of oral deposits. It may be one of the important factors responsible for higher prevalence and severity of periodontal disease in the Asiatic countries.

I.3.9.3 Effect of alcohol drinking

It is really a surprise to find that only one epidemiological study of periodontal disease has considered alcohol drinking as a possible etiological factor. Summers and Oberman studied 408 subjects 20 years old or more, consisting a 10% probability sample from Tecumseh, Michigan. Correlation analysis was made between periodontal disease, measured as PDI and 12 intrinsic variables including alcohol consumption amount. Results showed no significant correlation between weekly alcohol consumption amount and PDI score either with or without holding age constant (430). Further analysis of the data indicated no statistical significant difference of weekly consumption amount of alcohol among three groups of PDI gradient and edentulous subjects (431). More studies are needed before any generalization can be made from this study.

In summary, cigarette smoking tends to increase the prevalence and severity of both acute and chronic periodontal disease, by possibly more accumulation of dental deposits. Betel nut chewing can have even greater detrimental effects than smoking on periodontal tissues. There is not enough studies investigating the effect of alcohol drinking on periodontal tissues, though the data available seem to indicate little, if any, relationship between these two variables.

I.3.10 Association with dental caries

There is a commonly held opinion that periodontal disease is inversely associated with dental caries. In the early fifties of this century, some extensive reviews and discussions were directed to this problem (32,216,271). Kesel concluded from clinical reports and laboratory studies that the weight of evidence pointed to an antagonism between periodontal disease and dental decay (216). Marshall-Day believed from epidemiological data that these two diseases varied independently of each other (271). And Biddy stated that data based upon epidemiological studies, physiological, systemic as well as local factors did not give clear cut evidence indicating the antagonistic relationship between these two dental disease (32).

Cross-sectional epidemiological data suggest that some demographic factors provide indirect evidences that dental caries and periodontal disease do not have similar distributions. Factors like sex (202,258,291), socio-economical factors (212, 214,270), race (202,214), culture (172), local geographic distribution (367), world distribution (270,271,363,368), as well as war environment (220) are commonly correlated with dental caries and periodontal disease in an opposite direction as far as a population is concerned, which has been taken as evidences of antagonistic relationship between these two diseases. In addition to this, the distinct differences in prevalence according to age of subjects (32,272,291) and morphological types of teeth (222,292,470) between caries and periodontal disease also suggest that these two disease are unlikely caused by the same agents.

Untreated caries seems to be positively associated with more gingivitis and PI score, whereas filled teeth are inversely associated with gingivitis and PI score (280,366,399,470). Although study made by Barros and Witkop demonstrate a significantly positive corelation between DMF-teeth and PI score as well as GR score (78). Other studies do not support significant relationship between DMF-teeth and PI score (103,279), DMF-teeth and prevalence of gingivitis (280) or between DMF-teeth or surface and the severity of gingivitis (276,277).

As mentioned previously that active periods between caries and periodonatl disease differ distinctly (32,272,291), a cross-sectional study may not reveal a true relationship of disease activity or incidence between these two diseases. To demonstrate the possible fallacy which may result when a cross-sectional study method is employed to correlate two chronic diseases, such as dental caries and periodontal disease, Russell conducted a one-year longitudinal study on 506 school children, aged 13 to 14 years. From cross-sectional data, four correlation co-efficients of total DMF-teeth with PI scores in 13- and 14- year-old boys and girls range from --- 0.24 to +0.57 which do not allow any consistent conclusion. But when mean PI scores at the beginning and the end of one year are correlated with mean numbers of new DMF-teeth, it becomes clear that for children with simple gingivitis only or PI scores below 0.75, PI scores are correlated positively with new DMF-teeth, caries activity is, however, at a near standstill in children with advanced destructive stages of periodontal disease or PI scores above 0.80 (357).

Thus, the research data so far fail to demonstrate any simple clear cut relation between caries and periodontal disease. Neither the commonly held opinion that these two dental diseases are essentially the diseases of dental plaque is supported. Quite contrary, research data suggest that these two dental diseases have distinct epidemiologic patterns and do not share many common disease related variables, and that their association may vary from one disease stage to another, from age to age and depend on particular human environments. In fact, the definite beneficial effect of fluoride intake to reduction of dental caries and lack of such effect on periodontal disease also implies that the prevalences of these two dental diseases are rather independent one from another.

I.3.11 Malocclusion, trauma from occlusion and oral habits

I.3.11.1 Malocclusion

Malocclusion of teeth has long been considered by clinicians a likely causative factor in disease of the supporting structures of the tooth. But there are few reports available in literature that can supply quantitative data to support them. Ditto and Hall studied on 143 periodontal patients and reported that Angle's classification of malocclusion (5) did not affect the percentage distribution of cases in the periodontal classification (88). On the other hand, studies made by Miller and Hobson on school children (290) and by Geiger on 188 consecutive cases on periodontal disease (123) revealed that subjects with normal occlusion had milder periodontal disease than those who belonged to one of three Angle's classifications. Except Geiger's data (123), tooth crowding has been found to correlate positively with prevalence of periodontal disease (88), gingivitis (277,280,290,351,374), and PI scores (33,324). Beagrie and James (21) reported no significant relationship between modified Russell's PI index score (Galloway index (122) and either axial inclination measured from radiograph or malalignment index designed by Van Kirk and Rennell (449). Neither did Wade find a significant association between gingivitis and tooth alignment from his study on British and Iraqui children (454). Suomi reported, from a population study, that the degree of contact of two neighboring teeth insignificantly associated with the pocket depth at interproximal surfaces (432). The relationship between periodontal disease and overbite or overjet is not very consistent either. It is, however, possibly resulted from the facts that those studies which come out negative findings (88, 123) do not employ quantitative measurements of periodontal disease, overbite or overjet, nor hold age in constance when comparisons are made. With severity scales, PI score (33,324) and prevalence of gingivitis (280) are found to be significantly correlated with degree of overjet and overbite. Following the introduction of quantitative indices of malocclusion, such as Occlucal Feature Index (OFI) by U.S. National Institute of Health, Dental Research (324), Handicapping Labie-Lingual Deviations (HLD) by Draker (89) and maloccluded teeth counts by Massler and Frankel (275), these indices have been found to be correlated significantly with PMA index (277,351) and PI score (33,324).

Thus, it seems very likely that the degree of malocclusion correlate with severity of periodontal disease, though the strength of the association varies from one feature of malocclusion to another. One must also bear in mind that most malocclusion indices are designed for the purpose of orthodontic treatment and, therefore, the weights assigned to various component features within each index may not be much meaningful in terms of their relative importance in the pathogenesis of periodontal disease.

I.3.11.2 Trauma from occlusion

Karolzi in 1901 suggested that excessive occlusal forces lead to pyorrhea(206). That statement is still a controversial issue today. Trauma from occlusion is defined by Glickman as " the injury to periodontal tissue caused by occlusal forces (138). Since the introduction of a concept by Glickman and Smulow (142) and by Glickman alone (137,139,140), through experiments on the monkey, that inflammation and trauma from occlusion are co-destructive factors in chronic periodontal disease and that the excessive occlusal forces alter the pathway of gingival inflammation and constitute a significant factor in determining the pattern of bone destruction in periodontal disease, Glickman and Smulow have confirmed these findings in human autopsy materials (143,144),so has Ross supported the concept from his clinical observation (353). But recently, neither monkey's experiments made by Glickman and Smulow (145) and Comar <u>et al</u> (72), nor human biopsy material by Stahl (423) could demonstrate the progression of inflammatory response into periodontal ligament.

The possible explanation to foregoing contradictive findings is the poor agreement as to the criteria of trauma from occlusion. Studies on human subjects have employed one or combination of the parameters, such as increased mobility, various type of bone resorption, increased width of periodontal space, increased thickness of lamina dura and premature contact, as criteria of trauma from occlusion (143,144,353,423). However, these clinical and roentgenographic study, Posselt and Maunsback (323) reported that mobility from occlusion, as a diagnostic criteria of trauma from occlusion, seldom accompanied by roentgenographic signs and that roentgenographic signs were not always accompanied by mobility from occlusion. Whether mobility from occlusion is a correct criteria of trauma from occlusion remains a question. In a survey, Lovdal <u>et al</u> divided the subjects into two main groups: one having eight teeth or less and the other having more than eight teeth in contact during occlusion and chewing. Their results showed a significant higher gross tooth mobility in first group than in second group which was independent of the amounts of soft deposits, but there was no significant difference in alveolar bone resorption and pocket formation (256). Similar findings were reported by other investigators (304,325,326). Ringsdorf <u>et al</u> also failed to observed the difference in PMA scores of pregnant women between those who had 18 teeth or less and those of 22 teeth or over (344).

Thus, the best conclusion one can draw from these reports is that trauma from occlusion itself does not likely initiate periodontal disease and that it is really not clear whether trauma from occlusion can facilitate the progression of inflammation into periodontal ligament and, hence, cause pocket formation.

I.3.11.3 Oral habits

Tongue thrusting is a habit which entails the persistent wedging of the tongue against the teeth, especially in the anterior region. Carranga et al (46), Dechaume (85) and Sheppard (406) feel that tongue thrusting produces excessive lateral pressure which may be traumatic to the periodontium. Glickman (138) also reported that togue thrusting causes spreading and tilting of the anterior teeth, with an open bite, anteriorly, posteriorly, or in premolar area. The antagonism between forces that direct the teeth labially, and inward pressure from the lip, may lead to tooth mobility (138). All these reports are based on clinical case observations, and, so far, no quantitative data are available.

Bruxism consists of aggressive, repetitive or continuous grinding or gritting of the teeth during the day or night or both. Clenching or clamping entails continued, intermittent or pulsating pressures, usually with the teeth intercuspated. And clicking or tapping of the teeth may be performed with the teeth in functional or abnormal positions (138). All these habits are sometimes refered to by the term "parafunction" which designates tooth contacts in activities other than chewing and swallowing (91).

Bruxism causes excessive tooth wear which is characterized by facets on tooth surfaces not ordinarily reached by functional movements and pronounced facets in normal functional areas. Frisch et al examined a group of 95 patients with varying degree of periodontal disease, and found that all of them revealed some faceting.(119). However, in a later study of 200 patients who had normal periodontal tissue, they, again, found varying degree of faceting (118). Their results, therefore, tend to negate the belief that the presence of facets necessarily accompanies with periodontal disease. Studies made by Belting and his co-workers on psychiatric patients and non-psychiatric patient (24) and on diabetic patients and their controls (25) failed to reveal any correlation between bruxism and severity of periodontal disease. Baer and co-workers pointed out that occlusal wear did not in itself produce alveolar destruction (13). The periodontium in patient with bruxism often responds favorably to the increased function by thickening of the periodontal ligament and increased density of alveolar bone, and clenching and tapping habits which concentrate on isolated teeth or section of the arch are more likely to be injurious than the generalized grinding in bruxism (138). The suggestion, made by Ingle (179) and Leof (234), that bruxism and clamping could aggravate existing periodontal disease and lead to tooth mobility remains to be proved, or disproved, by a better study design other than clinical impressions.

Mouth breathing is another habit often associated with gingivitis (138,246). The gingival changes include erythema, edema, enlargement and a diffuse surface shininess in the exposed area. The maxillary anterior region is the common site of such involvement. Its harmful effect is generally attributed, without any substantial evidence, to irritation from surface dehydration. Since experimental study of simulated mouth breathing failed to support the dehydration theory (339), there is still much to be learned about the exact manner of production of the gingival reaction from mouth breathing.

To conclude, subjects having severe malocclusion tend to have more severe periodontal disease. Trauma from occlusion itself does not likely initiate the disease, and its role in progression of periodontal disease is uncertain. Mouth breathing is frequently associated with gingivitis of anterior teeth. There is a need for epidemiological studies about the effect of other oral habits on periodontal tissues.

I.3.12 Microorganism

It is only during last decade that a great deal of works have been directed to examine the role of microorganisms in the pathogenesis of periodontal disease in human. There are a few articles in which extensive reviews have been made regarding various microbiological aspects of periodontal disease (14,102, 125,250,381,418,422).

Evidences of bacterial role in the etiology of periodontal disease in human are substantial indeed. Acute necrotizing ulcerative gingivitis has been found to be suspectible to the action of local or systemic antibiotics (148,166,294,380). There are also reports of varying degree of prevention or reduction of non-specific gingivitis through mouth rinse or systemic administration of antibiotics (80,253,426,429). Since bacterial debris is almost entirely bacteria in nature (9) the remarkable correlation of bacterial debris with severity of gingivitis (10,312,397) suggests that bacterial bodies bear a direct relationship to the severity of gingival inflammation. Study conducted by Schei et al (375) further demonstrated the amount of bacterial mass on the tooth surface as a most important attributing factor to the alveolar bone loss, as well as to tooth mobility in their separate report (256). The most direct evidence, of course, comes from experiment such as by Loe, Theilade and Jensen (252) who showed that a group of 12 dental students developed accumulations of soft debris (plaque) and clinically observable inflammation of gingiva when all oral hygiene measures were withdrawn. The plaque material was removed following the reinstitution of oral hygiene measures, and the gingivitis subsided. While these results do not, in themselves, prove the initiating role of bacteria in periodontal disease, the fact that the change in microflora occurred before gingivitis was clinically diagnosed, may indicate that the microorganisms play a role in the initiation of periodontal inflammation (252). They later repeated the similar experiment and achieved the same results (441). Furthermore, in their later short-term study, antibiotics were effective in cutting down subclinical gingival inflammatory signs (253). To add additional evidence, pure cultures of certain human gingival crevicular organisms have been shown to initiate a periodontal syndrome in laboratory animals (127,128,398,419). Although these data did not prove the etiologic role of these organisms in periodontal disease conclusively, they did clearly demonstrate the potential of human oral organisms to cause periodontal destruction.

The research, so far, fails to find a single species or group of microorganisms which are responsible for the production of gingival inflammation (102,250,381,418). Indeed, a number of studies have shown no marked difference in the microbial composition of gingival debris obtained from healthy versus diseased gingiva (97,132,300,419,441). The only microbes which seem to be different between the flora of the gingival crevicular area of children in comparison with adults are Bacteriodes melaninogenicus (15,84,215) and certain spirochetes (84) and even the appearance of these two kinds in adult population seem to be more related to their specific living environments and nutritional requirements than potential to produce periodontal disease (15,418). Thus, the only significant alteration associated with periodontal disease appears to be one of the total quantity of bacteria present (300,397).

It seems apparent that plaque formation is the first step initiating the periodontal disease (418). Streptococci mutans isolated from human gingival crevice is reported to form dextran which glue the organism to the tooth surface and to other organisms of the same type (126,128,129,131). Jensen <u>et al</u> reported that despite the suppression of the Gram-positive flora and spirochetes from daily use of vancomycin mouth rinse, plaque still formed which consisted all Gram-negative organisms (200). Once the microorganisms have accumulated, it is not clear how they lead to the destruction of the periodontium. It has been suggested that bacterial products in gingival crevices, such as lytic enzymes (74,86,130,165,382,384), endotoxin (412) and other toxic metabolites (102,250,381,418), all of which have the capacities to destroy the periodontal tissue, may be responsible for periodontal destruction. But it is also true that most of these lytic enzymes can be produced by the gingival tissues themselves (29,31,120,146,337), and that no differences have been observed in the specific activities, on wet weight basis, of various enzymes in gingival crevice debris obtained from normal and periodontally involved patients (74,300). One must, however, also bear in mind that the total mass of organisms is larger in periodontally involved individual (397) and, therefore, more bacterial products which may be capable of destroying the periodontal tissues (250,300,397,418,419).

Hypersensitivity and allergic phenomena as a mechanism of periodontal disease has been postulated and some researchers have demonstrate antibodies in sera and gingiva and skin allergic reaction to specific oral bacterias (305,306,307,376). Unfortunately, there is little evidence that immunopathologic processes actually operate in naturally occurring human periodontal disease (124,418).

In fact, one might even wonder whether there is sound evidence that the inflammatory process in the gingiva is destructive at all (418).

It is probably safe to state that the mechanism by which bacterias induce destruction of the periodontal tissues is not known (418). So far, we have found that some agents and process, which are suspected of being significant in the etiology of periodontal disease in human, are present in gingival crevice and periodontal tissue, and can induce the periodontal disease in experimental model systems. But we fail to demonstrate that these agents and process actually have an effect on the periodontal tissues in the course of human periodontal disease.

Despite the fact that transmissibility of periodontal syndrome in experimental animals has been demonstrated (87,205,217), nothing of this nature in human periodontal disease has yet been reported.

To summarize, there is strong evidence that oral bacterias are quantitatively associated with gingival inflammation. Studies so far fail to demonstrate any single species or a group of microorganisms which are responsible for gingival inflammation.

The mechanisms by which bacterias cause destruction of the periodontal tissues is not yet understood. Whether or not the periodontal disease is transmissible through bacterias from one person to another remains to be answered. There is no single study so far to investigate the possible role of virus in the prevalence or severity of periodontal disease.

I.3.13 Iatrogenic (Faulty dentistry)

The harmful effects created by professional dental care such as operative and prosthetic dentistry (302,347,473) and orthodontic treatment (333,466) have been studied and reviewed.

The single factor which has been most extensively studied is overhanging of the restoration. Clinical impression seems to indicate that overhanging restoration usually accompanies with gingival inflammation adjacent to it (138). Some clinical surveys substantiate such impression with statistical data (3,466,473) while others fail to demonstrate significant association between overhanging restoration and gingivitis (134, 432). The reason for the non-association finding in the study made by Gilmore and Sheiham (134) may be that a large percentage of overhangs may not have been in direct contact with the gingivae. There are two studies show that alveolar bone heights adjacent to overhangs are significantly lower than those of homologue teeth without such overhangs (34,134). One of these two studies also indicates that the more the overhang is in excess to gingival margin, in lower the alveolar bone height is as compared with that of the contralateral tooth (34).

Several reports have shown that gingival inflammation, mobility and bone destruction increase dramatically in teeth adjacent to partial denture.(44,45,111,386). In two well controlled clinical trials, the effect of removable splints on tooth mobility was investigated. Instead of reducing mobility, splinting led to increased mobility as compared with patients who received ordinary local hygienic treatment (334,474). These findings certainly can easily be criticized in terms of the desirability of the constructed removable partial dentures and splints. On the other hand, more research works are urgently needed to clarify this dilemma.

Other faulty restorations, such as failure to reproduce the contour of the buccal surfaces, inadequate or improperly located proximal contacts, failure to re-establish adquate interproximal embrasures and failure of the restoration to conform with occlusal wear patterns have all been mentioned as common causes of gingivitis and periodontal disease (138,302,347), but very few studies are available to support these opinions.

After a series of studies about tissue reaction to artificial crowns (455), rough surfaces (456), zinc phosphate cement fillings (457), gold foil (458), and metal wires (459). Waerhaug concluded later that subgingival restorations were among the major factors in periodontal disease (460), which was not resulted from mechanical or chemical irritations of filling materials, but was caused by the bacterial plaque which formed in spaces between the crown and the tooth preparation. Since the bacterial plaque in these areas can not be removed completely, and some of it will be left in those space and from these retaining microorganisms the apical proliferation of the plaque will occur. Thus, Waerhaug suggested that it was better to finish the restoration above the gingival margin whenever perfect adapted artificial crown was not attainable (460).

To summarize, although numerous kinds of faulty dental restorations and orthodontic appliances have been condemned for various stages of periodontal disease, very few research had been done to clarify the exact nature. However, overhanging restorations, unsatisfactorily constructed partial dentures and removable splints seem likily to have detrimental effect on periodontal tissues either through bacterial accumulation, faulty force on teeth or both.

I.4 Existing Indices used for studies of periodontal disease

I.4.1 Indices to estimate the alternation of the periodontal tissues

The high prevalence of periodontal disease almost in every population makes comparison of prevalence of the disease between populations very little meaning. Therefore, several scoring methods or indices have been developed which takes stages of the periodontal disease into consideration. The measurement of periodontal disease is much more difficulty than that of dental caries due to three facts; 1) judgement of gingivitis severity is very subjective and the inflammation is reversible. The alveolar bone can not be directly observed without radiograph. Its resorption is chronic in nature, and yet the best measure of periodontal tissue damage. 2) to transfer qualitative data into numeric index involves arbitrary assignment of the gradient scores. It is especially difficult when one has to combine both gingival inflammation and alveolar bone destruction into a single index. Because no one has proved that gingival inflammation actually progresses to periodontitis or alveolar bone loss (155,418). 3) there is no way to deal with missing teeth, because lack of history about the causes of missing or extraction.

It is commonly believed from clinical observation that gingivitis per se can recover to healthy and origingal condition, whereas gingival recession, apical migration of epithelial attachment and resorption of alveolar bone can not recover to original tissue state. Therefore, indices for periodontal disease have been arbitrary classified into irreversible and composite indices (462).

I.4.1.1 "Irreversible" indices

I.4.1.1.1 Indices of alveolar bone loss

Indices measuring alveolar bone loss are possibly the earliest periodontal disease indices for epidemiological studies. Both Sheppard (404) and Marshall-Day and Shouri (272) used it for studying hospitalized patients and tried to measure to one-tenth of the root length. Later, Sandler and Stahl (371) and Cohen et al (368) assigned 4 or 5 point-scale respectively for the proportion of bone loss. To refine the scale, Schei et al (375) placed a translucent ruler on the top of the radiograph, by this device the length of the root is divided into 10 equally long areas, and it was possible to assess the height of bone or bone loss with an accuracy of 1/20. Most recently, Bjorn et al (35) designed a ruler which had 20 divisions or radii, and they assessed the marginal bone loss by representing it as proportion of the alveolar bone of the total tooth instead of root length.

Although radiographic assessment of alveolar bone loss appear to be the most objective method, it has many shortcomings: 1) very high percentage which are not measurable for various radiographic reasons (35,375). 2) difficulty to standardize radiographic technique, especially angulation of x-ray cone to the teeth. 3) the index gives no information about the condition of soft tissue or the presence or absence of active disease. 4) it is time consuming and requires heavy equipment.

I.4.1.1.2 Gingival Recession Index

This index is considered irreversible because it is believed that gingival recession to the extent of cementum exposure is associated with some resorption of alveolar bone (424). The index represents the percentage of teeth in which the cemento-enamel junction is exposed (371,424). It takes very little time to score it for one individual. It is, however, a very crude index because it does not differentiate slight versus marked recession. Still the biggest drawback of this index is the frequent lack of association between presence or absence of recession and activity or even severity of the overall periodontal disease. When used together with other reversible indices on adult population, it has been shown to give additional information (17,18,247,367,368).

I.4.1.1.3 Level of epithelial attachment

This parameter has recently been taken as a separate measurement for the destructive phase of the periodontal disease (432, 433). It requires two intermediate measurements, namely, the distance from gingival margin to cemento-enamel junction and the distance from gingival margin to epithelial attachment level.

The algebraic difference of these two intermediate measurements is the level of epithelial attachment in relation to cemento-enamel junction, or epithelial detachment level. Its reproducibility is only recently subjected to serious evaluation both by present author in this thesis and by Suomi et al (433). Owing to its slow progression, it shares some disadvantages inherited in the index of alveolar bone loss, though it is less time consuming and does not need heavy equipment. Furthermore, it allows for assessing four surfaces of each tooth. Using this index together with gingival index is now under study (135,416,433,434).

I.4.1.2 "Reversible" indices

One class of reversible indices is exclusively concerned with the clinical signs of active gingival inflammation (gingival indices). Another class takes into account also the destructive phase by giving more weight to pocket deepening and bone resorption.

I.4.1.2.1 Gingival indices

The P-M-A index by Schour and Massler (378) is probably the first index which scores the active periodontal disease. The three letters stand for papillary, marginal and attached gingival unit. The originators postulated that the inflammation would start from the papillary gingiva and spread thereafter to the marginal gingiva and finally reach the attached gingiva. Therefore, P-M-A count will itself give some crude information about severity. Massler (274) later suggested to assign, to each gingival unit, various score for severity of inflammation. The application of P-M-A index has been changed many times and various modifications have been introduced (40,77,197,316,349,467). Early, Brown et al (40) and Rosenweig (349) applied different weights to P-M-A. Weisinger et al (467) proposed "the percentage of areas involved" as an alternative presentation. They further suggested giving 0-4 score for each gingival unit so as to obtain "the percentage of the highest possible P-M-A score." Three other studies (77,197,316) have taken both intensity of inflammation and anatomical gingival unit into consideration. In clinical test, gingivitis hardly occurs only on papilla without marginal involved (316), thus any weights giving to these three gingival units are very questionable. When severity scale is incorporated into original P-M-A index, it is hoped such refinement will increase sensitivity. Unfortunately, the criteria of severity involve a great deal of subjective judgement, that make calibration very difficult and reproducibility rather poor (462).

The Gingival Index as developed by Loe and Silness (251) has gained considerable acceptance. The index is based on the severity of inflammation rather than its extent. The severity scale runs from 0 to 3 and its criteria set up clean cut distinction between the presence or absence of gingivitis and, if present, the "tendency to bleed under several types of stimulation. The validity of the criteria was recently substantiated through exudate measurements and microscopic examination (314). But in the field application, the control of "probing" method may not be easy. Its reproducibility has not been extensively studied possibly handicapped by its nature of bleeding. Loe (249) claimed:"The reproducibility is good provided the examiner's knowledge of periodontal biology and pathology is optimal." Gingival Index designed by McHugh et al (282) is similar to that of Loe and Silness, except the formers select different teeth for assessment. Gingivitis Evalution System designed by Cheraskin and Ringsdorf (50) is rather subjective in criteria. DHC Index for Gingival Assessment as introduced by Suomi (432) and Suomi (433) is based on colour change and the extension of adjacent tissue. Its severity scale ranges from 0 to 2. Intraobbserver agreement with this index can improve greatly from 59% on the first day to 87% on the third day of field practice (416).

Recently, one clinico-laboratory method was developed by Klinkhamer (224) which measured the orogranulocytic migratory rate and was called OMR index. The OMR is found to reflex correlation between inflammation and vascularization of gingival and periodontal tissue (224). It seems very useful to detect subclinical gingivitis and minor change of inflammation. It also has one advantage in that only laboratory technician is needed. But the heavy laboratory set-up may limit is for field study. If the facilities are available, OMR index may be found very useful in evaluation for clinical trials relating minor change of gingival condition.

All gingival indices share one rather big shortcoming, that is, they have no provision for destructive phases of the periodontal disease. They have, therefore, very limited value when used alone in a population with whole range of age.

I.4.1.2.2 Periodontal indices

Russell's Periodontal Index (PI) (356) is the first attempt to consider both gingivitis and the "destructive consequency" of gingivitis in an index. It still is the only reversible index which consider both gingivitis and destructive phases of the disease. In essence, PI is a morbidity index which merely answers yes or no as to whether a tooth has gingivitis (score 1 for mild and 2 for severe gingivitis), gingivitis with pocket formation (score 6) or has lost its function due to periodontal destruction (score 8). It is reversible because the presence of true pocket, i.e., epithelial detachment, without co-existing gingivitis will be scored as 0. Therefore, it is theoretically suitable for clinical trial. In practice, however, unless the gingivitis disappears clinically following the trials, the criteria for gingivitis in PI is not as sensitive as other gingival indices. Russell's PI has been favored and extensively utilized in world population studies. It is simple to use. Since the most advanced phases of pathology, in which examiners agree best in judgement, are given the highest scores, it serves very well in comparisons of population scores. Indeed, its greatest value is the great volume of world prevalence information generated through its use (363,364,365,368). With strict calibration to the originator (363,364,365), PI is still the best one to serve this particular purpose. The greatest shortcoming of PI is that it has no provision for differentiation between a slight pocket deepening (score6) and one reaching to the apew (although in the latter case a score of 8 may usually be given), nor for more break-down of gingivitis severity. Further, its reproducibility for each measurement or even an average score for individual subject is rather unsatisfactory (82). For these reasons, PI should be consider unsuitable for small sample in a clinical trial or for other longitudinal studies in which both sensitivity and reproducibility are essential.

Lilienthal et al (242) selected 6 representative teeth and score them with the same criteria of Russell's PI, the results showed no bias in this method when compared with the original method in which all teeth are scored. Galloway (122) also modified Russell's PI in which he employed pocket estimation as a way to refine the PI score, but its additional validity has not been wholly studied, neither is it familiar to other researchers.

I.4.1.3 "Composite" indices

Periodontal Disease Index (PDI) introduced by Ramfjord (329) is, in principle, a further development of Russell's PI. It is particularly designed for assessing the extent of pocket deepening below the cemento-enamel junction. Only six selected, but well represented (199), teeth are measured. Gingivitis is scored on a scale ranging from zero through 3 according to presence or absence of gingivitis, and, if present, extension around the tooth, bleeding or ulceration. If there is loss of epithelial attachment for less than 3 mm., the gingival score is ignored, and a score of 4 is given. If the loss is between 3 and 6 mm., the score is 5, and the loss of attachment of more than 6 mm. is scored as 6. There is evidence that this measurement is as useful as radiographs in estimating the degree of alveolar bone loss. (225). Since gingivitis scores are ignored whenever epithelial detachment is observed, it follows without any doubt that PDI can not be used for a clinical trial in which gingival inflammatory is the major interest. It can also be questioned whether the arbitrary figures set for the different stages of disease and destruction give the correct weight to each condition. Furthermore, since epithelial detachment can be measured as millimeter in scale, there seems to be no real need for transforming measurements in millimeter to a different system of figures, the index (249). Finally, it is still a contraversial issue regarding the justification for combining gingivitis index and epithelial detachment into a composite index (249), because we do not know exactly whether or not the apical migration of epithelial attachment and alveolar bone resorption is the result of progression from gingivitis.

O'Leary et al (310) used the same criteria of Ramfjord's PDI, but divided the mouth into six segments and chose the tooth of highest score in each segment as segment score. They have shown that this method of selection provides information closer to whole mouth examination than if 6 teeth in Ramfjord's method are measured (308). Their method is called Gingival-Periodontal Index (GPI). Later Lightner et al (240) made slight modification of GPI of O'Leary et al regarding the definition of positive epithelial detachment. They also gave provision for the score of gingival recession similar to that of epithelial detachment. They suggested not to combine the gingival and periodontal scores into a single index. Gingival-Bone Count (GBcount) as designed by Dunning and Leach (94) shares the same philosophy, advantage and disadvantage of those from Ramfjord's PDI. Their bone score ranges from 0 to 5 and is judged according to the pocket depth or alveolar bone loss (from radiograph) reaching to various quarter root length. Their later study revealed limited additional information which was gained from radiograph following clinical assessment. Hence, they suggested that whole-mouth surveys of large populations are probably best made without use of any radiographs at all (95). Slome (415) also introduced a Periodontal Score System in which the scale ranged from 0 to 10. The criteria is mainly a combination of three elementary parameter, i.e., gingival unit involved, number of teeth involved in a segment and the quality of disease, such as gingivitis, epithelial detachment or loss of tooth function. Such cook-up involved prejudged pathogenetic importance of each condition. One can also predict that some clinical condition may not fall into any of the 11 categories. Periodontal Disease Rate (PDR) by Sandler and Stahl (372) classifies severe gingivitis as definite alveolar bone loss.

Of course, no one should expect to find an index which will serve all purposes. The foregoing indices reviewed have served their own particular needs to some extent. Russell's PI is adequate when population prevalences of periodontal disease are compared. GI from Loe and Silness seems suitable for the study of oral hygiene clinical trials. Ramfjord's PDI and GB count from Dunning and Leach can improve the sensitivity of Russell's PI. But, if one wants to make a longitudinal study which involves both gingival inflammation and destruction of underlying supporting tissue, it is obvious no index so far is appropriate due to the facts that its sensitivity and, especially, the reproducibility has not subject to serious and critical evaluation. The prognostic significance of each facet of tissue alteration which is the basis of giving weights or scores has never been validated.

I.4.2 Indices to estimate the nature and amount of oral irritants and deposits.

Corresponding to the development of periodontal indices and their applications in the field studies, various indices measuring the degree of oral deposits and irritants have been introduced (6,36,108,156,157,240,282,310,329,407,410,415,432,452,453).

One of the most extensively used in population studies is the Oral Hygiene Index (OHI) by Greene and Vermillion (156). This index was later modified by the same authors and became the Simplified Oral Hygiene Index (OHI-S). The OHI consists of Debris Index and Calculus Index (DI and CI), each of which ranges in scale from 0 to 3 depending on the coronal thirds covered by debris and/or calculus. With the OHI-S, only six selected teeth and one surface for each tooth are assessed (157).

In clinical studies, especially researches on gingivitis, Plaque Index (PLI) introduced by Silness and Loe (410) has been used quite satisfactorily. This index emphasizes the plaque within the gingival pocket and on gingival margin as greater pathogenetic importance than that on the coronal thirds. The PLI also ranges in scale from 0 to 3.

The Irritant Index designed by O'Leary et al (310) and its slight modification made by Lightner et al measures the amount of materia alba, calculus as well as overhanging restorations. Retension Index which was developed by Bjorby and Loe (36) takes caries into consideration in addition to calculus and imperfect margin of restorations.

The most significant development for these measurements is the further refinement of the measurements specifically for supragingival calculus (108,452,453) and subgingival calculus (433). The intra-observer agreement is fairly good for supragingival calculus, but not satisfactory for subgingival calculus measurement (416). Its exact contribution to clinical trials and other longitudinal studies of the periodontal disease remains to be seen.

To summarize, there are a few indices which measure the quality and quantity of oral deposit and irritants. Selection of an index will certainly depends on the type of study. In general, comparison between populations required fast and simple indices, such as OHI-S. For clinical trials and longitudinal studies of small size, more sensitive indices are necessary. As discussed in the preceding sections, it is not certain yet whether these indices have contributed to the knowledge of the etiology of the periodontal disease.

I.5 Need for longitudinal studies to assess the direct association between possible etiological factor or factors and the incidence and progressing of periodontal disease

From the foregoing review of literatures, it is clear that cross-sectional studies have provided precious information with regard to the distribution of periodontal disease according to age, sex, world-wide geographic distribution, and ethnic groups, and its correlation with income, education, occupation, urban-rural residential factor and other social and cultural factors. These informations have contributed a great deal to the understanding of the epidemiologic pattern of periodontal disease.

At the same time cross-sectional epidemiological studies have collected a pool of data which are contradictory to the traditionally held opinions regarding the importance of nutritional and dietary factors, systemic conditions and diseases, in the initiation and progression of periodontal disease. In most instances, they also contradict with the results from clinical trials with respect to good or ill effects of various nutrients on periodontal tissues. Most of these contradictions can be explained by three reasons: First, in cross-sectional epidemiological studies, an assumption is made that the status of periodontal disease and suspected etiological variables and their association at time of survey existed in the past and will still be valid in the future. Second, in cross-sectional studies, investigators have taken oral hygiene factor (in fact, oral deposits) as a complete independent stimulating factor, and whenever other variables are found to be significantly associated with the prevalence and severity of the periodontal disease, standardization of oral deposit score and age is made that result in disappearance of such significant association between those variables and periodontal condition. Yet, that oral deposits are independent of the sequela of the periodontal disease is a dangerous assumption. Third, enormous clinical knowledge comes from case reports and clinical studies which had been conducted before sciences of epidemiology and statistics were introduced to dental profession.

There, however, also certain types of data that can not be obtained from cross-sectional studies: 1) documentation of true disease incidence, 2) relationship between cause and effect and 3) documentation of the stages in the development and progression of a disease or natural history of the disease. It is paramount important to obtain incidence data for gingivitis and destruction of epithelial attachment respectively as well as various suspected factors at time of and prior to the disease initiation. Only then, any significant correlation of suspected variables with incidence of gingivitis or epithelial detachment are really meaningful. This concept also hold for the severity of the disease provided that the data are complete from the inception of the disease. Longitudinal studies may also answer other important questions, such as: 1. Whether more severe gingivitis eventually lead to destruction of epithelial attachment and alveolar bone resorption? 2. Exact relationship between dental caries and periodontal disease. Does that relationship changes at different age of life? This question is important because it will provides a good reference to the current dental researchers who believe that both dental caries and periodontal disease are diseases of dental plaque and, therefore, can be prevented by the same method. Quite apart from epidemiological aspect, longitudinal studies are also needed for evaluating the beneficial effect on supporting alveolar bone following aggressive treatments for chronic periodontal pocket. These treatments have been claimed to stop the progression of gingivitis and alveolar bone resorption, and yet, only its effect on gingivitis has been demonstrated.

For these longitudinal studies, a periodontal index which is designed specifically for such purpose is essential. The component parameters of this kind of index should have good sensitivity as well as reproducibility, and information about the limits and variations of these two characters must be available at hand. Furthermore, these component parameters should be weighted according to their relative prognostic importance from field study so that a single index can be formulated.

APPENDIX II

THE COMPUTATION FOR THE STATISTIC OF AVERAGE AMOUNT OF INFORMATION TRANSMITTED

The computation for the statistic of average amount of information transmitted is analogous to that for analysis of variance in quantitative data. Namely, it involves each cell frequency, marginal total and over-all total. Its actual calculation consists almost entirely of the addition of terms of the form $n(\log_2 n)$. The example chosen for illustrating the calculation is the inter-observer variation as presented in Fig. 3. The steps in the computation are as follows:

i. Add together the values of $n(\log_2 n)$ corresponding to each cell frequency to obtain:

$$a = 206.1 + 4.8 + 15.5 + 268.1 + 2.0 + 2.0 + 69.5 + 2.0 + 19.7 = 589.7$$

ii. Add together the values of $n(\log_2 n)$ corresponding to each marginal total of reference estimate to obtain:

$$b = 226.5 + 325.2 + 80.7 + 28.5 = 660.9$$

iii. Add together the values of $n(\log_2 n)$ corresponding to each marginal total of observer X estimate to obtain:

$$c = 247.1 + 303.6 + 02.2 + 19.7 = 662.6$$

iv. Find the $N(\log_2 N)$ of over-all total. In this example, $N = 126$, this gives $d = 879.1$.

v. Now calculate the statistic as:

$$(a - b - c + d) / N = 145.3/126 = 1.15 \text{ binary digits.}$$

The actual maximum information can be calculated as:

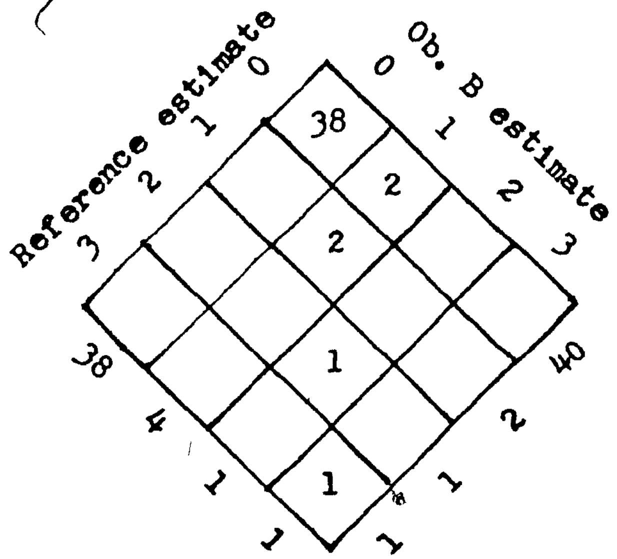
(d -b) / N = 218.2/126 = 1.73

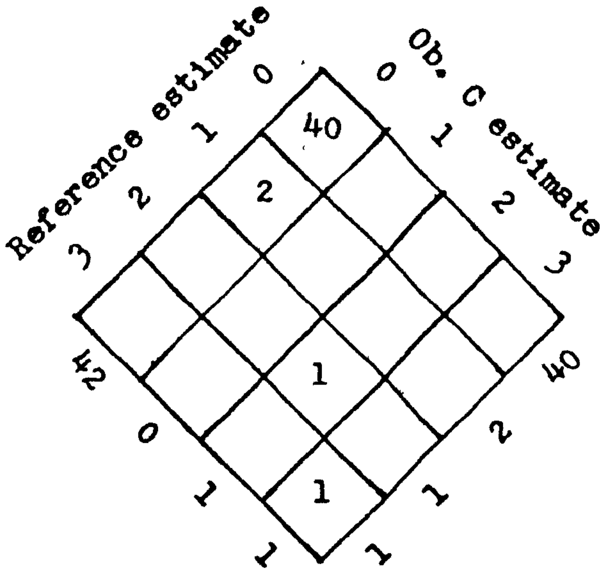
The information lost is calculated as:

(c - a) / N = 72.9/126 = 0.58

The proportion of information transmitted to the actual maximum information is: 1.15/1.73 = 66.5%

The statistic of average amount of information transmitted becomes less useful for a parameter which has very low prevalence rate or very unequal frequency distribution. The following two two-way distribution diagrams.showing the initial gingival bleeding estimates in 44 subjects according to observers B and C's estimates give the best example.





a = 203.4

b = 214.9

c = 207.4

d = 240.2

Maximum information: 0.57

Information transmitted: 0.48

Information lost: 0.09

a = 214.9

b = 214.9

c = 226.5

d = 240.2

Maximum information: 0.57

Information transmitted: 0.31

Information lost: 0.26

Based on the reference estimates, no gingival bleeding was observed in 40 subjects. Such frequency distribution results in two major disadvantsges; first, the actual maximum information available is very small amount due to little difference between the values of (d) and (b); second, the information lost would be little as long as the observer estimates do not result in any further increase of number to the group having the greatest frequency according to the reference estimates, otherwise, the information lost would be tremendous. The diagrams show that there were two cases in which the gingival bleeding was overestimated by observer B and underestimated by observer C. Thus, while the highest frequency according to the reference estimates is 40, the corresponding values for observers B and C's estimates are 38 and 42 respectively. Such difference results in very much greater value of (c) in the case of observer C estimates than in observer B estimates. Therefore, the information lost for observer C estimates (0.26 binary digits) is much greater than that for observer B estimates (0.09 binary digits).

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