PREVALENCE OF PERIODONTAL DISEASE AND ITS ASSOCIATION WITH TOBACCO USE: A COMMUNITY BASED STUDY IN THIRUVANANTHAPURAM DISTRICT, KERALA

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Dissertation submitted in partial fulfilment of the requirements for the award of the degree of Master of Public Health

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DECLARATION

I hereby certify that the work embodied in this dissertation entitled "Prevalence of periodontal disease and its association with tobacco use: A community based study in Thiruvananthapuram district, Kerala" is the result of original research and has not been submitted for any degree in any other University or Institution.

Thiruvananthapuram
19-5-2000

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CERTIFICATE

Certified that this dissertation entitled "Prevalence of periodontal disease and its association with tobacco use: A community based study in Thiruvananthapuram district, Kerala" is a record of bonafide original research work undertaken by Jayakrishnan. R in partial fulfilment of the requirements for the Degree of Master of Public Health, under our guidance and supervision.

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CONTENTS

ABSTRACT

Objectives: To study the prevalence and correlates of periodontal disease among adult population in Thiruvananthapuram district, Kerala. Periodontal disease is a risk factor for tooth loss and systemic diseases like cardiovascular disease. Prevalence of periodontal diseases is not reported from rural Kerala.

Design of the study: Cross-sectional survey using a cluster sampling technique.

Study setting: Six panchayaths from 3 community development blocks of Thiruvananthapuram district of Kerala.

Participants for the study: 504 individuals in the age group 30-50 years (214 F, 290 M), mean age 38.8 ± 7.1.

Intervention: Measurement of periodontal pockets by a dentist using the WHO index of Community Periodontal Index of Treatment Needs (CPITN)

Main outcome measures: Prevalence of periodontal disease (CPITN 3 & 4)

Results: The overall prevalence of periodontal disease was 63.5%(95%CI: 61.1-69.4). From the bivariate analysis the important correlates of periodontal disease identified were age group, sex, socio-economic status, oral hygiene habits, smoking and alcohol use. Multiple logistic regression analysis identified age groups 45-50(OR=5.48; 95%CI 3.01,9.96), socio-economic status (OR=1.84 95%CI 1.01-3.37) and tobacco smoking (OR=2.65 95%CI 1.29-5.41) as important correlates of periodontal disease.

Conclusion: In our community based study, around two thirds of the adult population had periodontal disease. The emergence of smoking as a risk factor for periodontal disease should be dealt with utmost importance. Educational efforts targeted to reduce smoking should be considered at the community level. Dental checkups should be started in an early age considering the progressive nature of the disease with age.

1. INTRODUCTION

1.1 The back ground

Periodontal disease afflicts many individuals at some point during life and is a major cause of tooth loss in adults. The distribution of periodontitis (inflammation of the periodontium) in the population suggest that a subset of individuals is highly susceptible to this infection, while remaining majority exhibit varying degrees of resistance and moderate susceptibility. Current data indicate that dental plaque is the cause of periodontitis and cigarette smoking is a known risk factor, which adversely influences periodontal health. There is evidence that cigarette smoking exerts both systemic and local effects. Though studies had shown an association between the two, precise mechanisms underlying the effects of smoking on periodontal status and wound healing remain unresolved.

The disease is initially confined to the gingiva and is called chronic marginal gingivitis; later the supporting tissues are involved, and are called marginal periodontitis. Gingivitis is a problem frequently occurring in young adults, which often gets unnoticed. Periodontitis or inflammation of the periodontium results from the extension of the inflammatory process initiated in the gingiva to the supporting periodontal tissues in which case there is bone damage and loosening and eventual loss of teeth. (For details please refer appendix 1)

Epidemiological surveys conducted throughout the world point to the universal distribution of dental caries and periodontal diseases.

1.2 The problem

The two most common diseases affecting the oral cavity are dental caries (tooth decay) and periodontal disease.[1] (Periodontal ligament is the connective tissue attaching the tooth to the alveolar bone.) Dental caries are more commonly found in children and young adults because of the consumption of large amount of carbohydrates in the form of sucrose. This becomes the main source of acid for demineralization when microorganisms have acted upon them.

At one time it was assumed that dental caries and periodontal diseases were associated with the life styles of persons living in more technologically advanced societies, where soft, rich foods are eaten regularly, providing materials toward the formation of plaque and support of the bacteria in the plaque. But recent investigations show that people in the less developed nations, who are relatively free of tooth decay, eventually develop periodontal disease. Modern periodontal epidemiology rediscovered focal infections and indicated that the general health has a crucial impact on the periodontal health and periodontal disease has also a major impact on the general health status of the patient.[2]

While a significant proportion of the population is susceptible to periodontitis it appears that there is a large segment of population that is not susceptible to the severe forms of periodontitis. This observation leads to the fact that there are risk factors that modulate susceptibility or resistance to destructive periodontitis.

A large body of epidemiological research indicates that tobacco use has deleterious effects producing a wide spectrum of diseases including cancers of the mouth, lung, larynx, pharynx; diseases of the heart, circulatory system and lungs; and if used during pregnancy, produces adverse effects on the fetus. There is a growing body of scientific evidence indicating that nicotine contributes to the progression of periodontal disease, and is detrimental to healing following periodontal therapy. Nicotine has toxic effects on peripheral circulation, which causes gingival vasoconstriction. As a result a decreased number of immune cells are available in the gingival tissue, which translates into a weakened defense-reparative system.[3] Evidence suggests that smoking is consistently associated with poorer levels of oral hygiene and consequently smokers have increased accumulation of plaque and calculus.[4] A strong association has been shown between periodontal disease and coronary heart diseases, as well as between periodontal disease and cerebrovascular diseases (stroke). The subgingival microflora and the continuous latent bacteremia (presence of viable bacteria in the circulating blood) and endotoxemia (bacterial toxin in the blood) originated from the periodontal pockets might be responsible for the damage of the vascular endothelial integrity, platelet functions and blood coagulation.[5] Though studies in the last two decades have shown a positive correlation between tobacco use and periodontal diseases, public awareness regarding the severity of periodontal disease and its complications is very minimal. As stated above this can be a warning sign for other systemic diseases and can also act as a 'foci' for infections. Tobacco products can alter normal host responses to neutralize infections and can also stimulate pathologic micro-organisms to destroy surrounding tissues.

Several studies have been done on the effect of tobacco smoking on the periodontium and have found an association between the two but limited studies were conducted to correlate tobacco chewing and periodontal disease. The importance of this study is to assess the prevalence of periodontal disease and also to compare the periodontal disease status among tobacco users and non-users.

1.3 Objectives

The objectives of this study were

1.To estimate the prevalence of periodontal disease among adult population in Thiruvananthapuram district, Kerala.

2.To compare the periodontal disease status among tobacco smokers, chewers and nonusers.

2. REVIEW OF LITERATURE

Kerr NW[6] studied the prevalence and natural history of periodontal disease in medieval Scotland (900-1600 AD) by examining the characteristics of interdental and crestal bone of jawbones obtained from archeological excavation. No individual over 11 years had a healthy periodontium. While gingivitis was widespread in the younger age groups, it was essentially a contained gingivitis, which slowly and constantly progressed to periodontitis. The pattern of prevalence and distribution of gingivitis and periodontitis appeared to be similar to that in modern civilizations. Massive amounts of supra gingival calculus were commonly found. The dentition start to disintegrate at an early age with most teeth virtually exfoliating after the age of about 40 years.

2.1. Tobacco use and its effect on oral structures

Tobacco produces a tenacious dark brown or black surface deposit with brown discoloration on the tooth surface. Staining is not necessarily proportional to the amount of tobacco consumed, but depends to a considerable degree on pre-existent coatings, which attach the tobacco products to the tooth surface[7].

Christen AG et al (1970)[8] reported that tobacco use can cause excessive abrasion of the incisal and occlusal surfaces of the teeth and periodontal disease when accompanied by poor oral hygiene, neglect, and an unfavorable systemic back ground, sinusitis, delayed wound healing, altered taste and smell, hairy tongue and leukoplakia.

Anil S, Hari S, Vijayakumar T (1990)[9] in a study conducted in the corporation and Municipality areas of Trivandrum district reported that deep periodontal pockets of more than 6mm were found in 33% of subjects and shallow pockets were found in 77% of the subjects of the age group 35-44 years. The mean number of sextants by score per person also showed the progression of periodontal diseases with advancing age.

2.2. The effects of tobacco smoking on the prevalence and severity of periodontal disease

Cigarette smoking has long been suspected to be associated with a variety of oral conditions including periodontal diseases. It is associated with an increased disease rate in terms of periodontal bone loss, periodontal attachment loss, as well as periodontal pocket formation. In addition, it exerts a masking effect on gingival symptoms of inflammation. Experimental evidence accumulated over the last two decades has indicated that cigarette smoking is probably a true risk factor for periodontitis. Smokers have both increased prevalence and more severe extent of periodontal diseases as well as higher prevalence of tooth loss and edentulism, compared to nonsmokers.

Preber and Bergstrom(1986)[10] surveyed the smoking habits in a population of Stockholm in 369 adult patients with moderate to severe periodontitis. The result showed that the frequency of daily cigarette smokers was significantly greater in the periodontitis sample. The odds ratio for a smoker to appear among periodontitis patients was more than double as compared to the population at large. Long-term studies have pointed out that smoking

was associated with recurrence of periodontitis during periodontal maintenance and the effect appeared to be dose dependent with heavy smokers presenting with higher levels of disease progression.

The indication that previous smokers have lower levels of risk of periodontitis compared to current smokers is considered to be the strongest available evidence that smoking cessation will result in an improved periodontal health. (Tonetti MS 1998)[11].

The early studies that examined the relation between smoking and oral cleanliness consistently found that smokers had poorer oral hygiene than nonsmoker. Ainamo(1971) considered that smokers did brush their teeth less frequently than non smokers, but Rajalo et al (1980) found only a weak association between smoking and tooth brushing[12].The increased level of debris observed in smokers have been tentatively attributed to

a) Personality traits leading to decreased oral hygiene habits.
b) Increased rate of plaque formation
c) Combination of above.

(Taani 1997)[13] studied the association between cigarette smoking and periodontal heath and found that oral hygiene was poor in both smokers and non-smokers. However, plaque and calculus scores were significantly higher in smokers than in non-smokers and no significant difference was found between the two groups with regard to gingival bleeding on probing or shallow or deep pocketing.

Weinstein et al 1996)[14] reported that smokers have more plaque than non smokers, even if no difference in the bacteria composition was found between smokers and non smokers samples, and the vascularity associated with plaque induced gingivitis is suppressed in smokers.

Sachit Anand Arora(1990)[15] in an epidemiological study conducted in Karnataka, India reported that smoking tobacco was found to have a more severe detrimental effect on the periodontium than tobacco chewers. It was also found that the severity of destruction had increased with increased consumption of cigarettes and tobacco chewed per day.

(Jeette et al 1993)[16] in their study reported that years of exposure to tobacco products was a statistically significant risk factor for tooth loss, coronal and root caries and periodontal diseases regardless of other social and behavioral factors. They concluded that life long tobacco use is a modifiable risk factor for poor dental health among older adults.

Arno et al (1958)[17] reported a significant association between tobacco consumption and gingivitis in a survey of 1346 predominantly male employees in a manufacturing company most of whom were aged 25-55 years. Severity of gingivitis increased with increasing tobacco consumption independent of age and oral hygiene status. The investigators did not discuss their findings in respect to the relation between smoking and oral hygiene in their report, but interestingly statistical analysis of their raw data, subjective though they are, indicated that smokers have poorer oral hygiene than non-smokers in individuals aged less than 45 years.

In the evaluation of a representative sample of the US, as part of the National health and Nutrition examination surveys, evaluated the relation ship of smoking to periodontal disease in 3000 individuals. In the study, smokers had significantly higher mean periodontal index scores in all age groups (25-75) years and significantly higher score in debris, calculus and oral hygiene indices. The data suggest that smoking has an independent direct association with periodontal disease.

In a study conducted by Bergstrom et al 1991[18] to find the association between cigarette smoking and periodontal bone loss, the study group consisted of 210 Swedish dental hygienist in the age group 24-60 years old. The study was based on bitewing radiographs. All subjects exhibited a high standard of oral hygiene, with no difference between smokers and non-smokers. Bone loss was significantly higher in smokers compared with non-smokers. The design of this study offers the advantage of homogeneity of the subject group in age, sex, education and socio economic status and found smoking as a major risk factor for periodontitis.

Results of the investigation exploring the association of smoking exposure with early-onset forms of periodontitis indicate that the adolescents who smoke had significantly elevated risk for generalized forms of juvenile periodontitis. The effect of smoking on the extent and prevalence of periodontal destruction has also shown to be dose dependent with estimates of total exposure to cigarette smoking being the most widely used measure of dose[19].

A higher risk for the development of oral cancer was associated with heavy smoking and heavy drinking, but alcohol drinking and tobacco smoking are also correlated with other oral cavity disease. Tobacco is the most important factor for leukoplakia, but other diseases like ANUG, periodontitis, median rhomboid glossitis, chronic hyperplastic candidiasis and others are also correlated to cigarette smoking. It was also observed that smokers have more plaque than nonsmokers, which is an important factor for periodontal disease.

Cigarette smoking is a known risk factor for many diseases, and mounting evidence suggest that smoking adversely influences periodontal health[20,21] Recent data indicates that the effect of tobacco smoking is a direct one and not due to poor oral hygiene and increased plaque relative to never smokers[22,23] While these studies support the role of smoking as a risk factor, estimates of the risk associated with smoking and the potential risk associated with former smoking are less well defined.

2.3. Effects of smokeless tobacco on the teeth and periodontium

There are over thirty different types of tobacco chewing habits in India.[24] As a result of intensive health education and rigid legislation restricting smoking in the developed countries, the prevalence of smoking in these countries is declining. How ever this has resulted in an increase in the use of smokeless tobacco. The marketing of tobacco in 'sachets' known as panmasala with ghutka has fortified the tobacco consumption in South East Asian countries. Use of tobacco for chewing is the most important etiological factor for cancer and precancerous lesions in the oral cavity.

The most consistently reported periodontal effects of smokeless tobacco are mucosal lesions that may also affect the gingiva, gingival recession, and various forms of periodontal diseases.[25]

Christen and colleagues (1970)[26] have reported that apart from oral cancer smokeless tobacco can produce significant effects on the hard tissues of the oral cavity in adults including discoloration of the teeth, fillings and abrasion of incisal and occlusal surfaces of the teeth. They also reported decreased ability to taste and smell and advanced periodontal disease. The oral problems other than leukoplakia that are frequently reported among smokeless tobacco users include gingival recession and gingivitis[27].

William HBowles etal(1995)[28] reported that tobacco products are known to cause oral soft tissue lesions, but they may also directly affect the teeth. They studied tobacco samples from sixteen brands of cigars, eight brands of snuff, four brands of chewing tobacco and several un processed tobacco leaves used as cigar wrappers. Insoluble particulate matter made up about 0.5 percent of the weight of an average tobacco sample, which is sufficient to produce abrasion of the teeth. In one study of several brands of chewing tobacco, the sucrose content averaged 15 percent. Chewing tobacco extract has also been found to enhance the growth of two forms of streptococci implicated in the production of dental caries[29] .

Offenbacher and Weathers(1985)[30] investigated the use of smokeless tobacco products, such as snuff and chewing tobacco, in a school population of 565 males with a mean age of 13.8 years. The overall prevalence of smokeless tobacco usage was 13.3%, which was much higher than the 1.4% prevalence of cigarette smoking. Pair wise chi-square analysis

revealed that there was no relation ship between smokeless tobacco usage and the prevalence of gingivitis, but that the prevalence of gingival recession was significantly elevated in smokeless tobacco users. They reported that smokeless tobacco usage was a significant risk factor in individuals with co-existing gingivitis, associated with a marked increase in the prevalence of gingival recession, mucosal pathology and caries experience. However those with clean mouths that were free of gingivitis, the use of smokeless tobacco was not associated with a change in the prevalence of gingival recession, mucosal pathology, or in the mean DMF score.

Shannon and Trodahl (1978)[31] concluded that it is likely, though not proved conclusively, that intra-oral use of tobacco reduces susceptibility to dental caries. They speculated that the relative freedom of caries reported in heavy tobacco chewers may be largely caused by accelerated salivary flow. The flow causes a physical cleaning action and a biochemical action, for example the bicarbonate content, pH and buffering capacity are all greatly increased at higher rates of salivation. Tobacco chewers and snuff dippers have a higher prevalence of excessive abrasion of the incisal and occlusal surface of the teeth. This is caused by the high level of abrasive grit inherent to tobacco products.

Although several studies have examined the relationship of smokeless tobacco usage on mucosal and dental changes the reports concerning the association between smokeless tobacco usage and periodontal health have been limited.

Christen et al(1979)[32] in their study "Intraoral leukoplakia, abrasion, periodontal break down and tooth loss in a snuff dipper" reported gingival recession and advanced

periodontal destruction, adjacent to the mucolabial region, which showed leukoplakia, a common sequela of tobacco use. They also reported a high prevalence of abrasion on occlusal and incisal surfaces of the teeth.

Van Wyk (1966)[33] made a clinicopathological study of 25 patients in South Africa who were confirmed snuff dippers. He reported that it was common to find gingival recession in teeth adjacent to areas in which tobacco is held. After histological examination he concluded that the necks of the teeth became exposed because of the recession of the gingiva than necrosis of the gingival tissues.

Pathak J.N Boghani C.P(1979)[34] did a clinical analysis to evaluate the role of tobacco chewing in periodontal destruction. In this study 731 subjects having various degrees of tobacco chewing habits were examined for their oral hygiene status, gingival and periodontal status. The results obtained were compared with 165 control subjects, who were apparently not having any habit. Wear pattern of teeth, gingival recession and mobility were also recorded and compared.

A) Oral hygiene status significantly deteriorated in persons having oral habits as compared to the controls.

B) It was statistically proved that as the duration and frequency of habit increases, the severity of gingival and periodontal destruction is also increased.

C) There was a significant correlation between the degree of habit and wear pattern of the teeth.

3. MATERIALS AND METHODS

As mentioned above the aims of the study are to assess the prevalence of periodontal disease and to assess the association between periodontal disease and tobacco use among adult population in Thiruvananthapuram district, Kerala.

3.1. Variables

The variables for the study include

1. Age group-those people in the age group of 30-50 years (tobacco users and non users)
2. Tobacco consumption in the form of smoking and chewing.
3. Sex- both men and woman in the age group 30-50 years were included in the study.
4. Other variables include household income, education, occupation, and alcohol use and oral hygiene habits.
5. Periodontal disease- CPITN grades 3 and 4 (outcome variable) (refer appendix 4)

Subjects were not included in the study if they were suffering from any systemic disease and were undergoing treatment.

3.2. Research hypothesis

Prevalence of Periodontal disease is more among tobacco users compared to nonusers.

3.3. Research methodology

3.3.1. Study sites

Thiruvananthapuram district was selected for the study because the researcher is a native of Thiruvananthapuram and is familiar to the study area. Other factors like close accessibility to the institute, feasibility of doing the study within the limited time period and the cost involved also influenced the selection of the area.

Thiruvananthapuram district has 11 community developmental blocks and these blocks were further categorised, based on their geographical locations, as high lands, midlands and low lands. Vellanad, Nedumangad and Perumkadavila developmental blocks were classified under the high land category. Kilimanoor, Vamanapuram and Nemom developmental blocks were placed in the midland category followed by Varkala, Chirayankeezhu, Kazhakuttam, Parassala and Athiyannoor being placed in the low land category. One community developmental block was randomly selected from each regions of the district. All three community developmental blocks namely Vellanad, Vamanapuram and Chirayankeezhu represent high land, mid land and low land. Seven to eight panchayats constitutes one community developmental block. . Each panchayat is again divided into wards. Two panchayats from each block was randomly selected for the survey. Vellanad and Poovachal panchayats were selected from Vellanad developmental block. Chirayankeezhu and kizhuvilam from Chirayankeezhu block and Vamanapuram and Pulimath panchayats were selected from Vamanapuram developmental block. All the wards from each selected panchayat were taken for the study. Thus a total of 66 wards were selected representing 66 clusters for the study.

3.3.2. Sample size estimation

Sample size was estimated using the following formula

$(Z^2 PQ/\Delta^2)^d$

Where,

Z- Confidence limit factor, which is 1.96 for 95% confidence interval

P-Prevalence of periodontal disease, which have been taken as 75%

Q=(1-P) i.e., the chance of non-occurrence

Δ - Precision factor (difference between assumed prevalence and worst expected prevalence)

d-design effect. Here it is taken as 1.5

From a study conducted by Anil S et al (1990)[9] to assess the periodontal conditions of a selected population in Trivandrum district it was found that of the 35-44 age group, 75% of the population were having periodontal pockets. With a 75% prevalence rate and to get a 95% confidence interval within ± 5%, the required sample size is 288. In this study the researcher opted for a cluster sampling method. Assuming a designed effect of 1.5 for cluster sampling, the minimum sample size required was estimated as 434. One cluster was taken from each ward and each cluster consisted of 7-10 people depending on the number of wards in each panchayath.

In certain wards there were houses with 3 to4 people in the 30-50 age group, and those people were also included in the study and thus the cluster size was not uniform.

A house-to-house survey was conducted in each ward so as to get the required number of subjects to form a cluster. Altogether 290 males and 214 females in the age group 30-50 years participated in the study, forming a total sample size of 504. (mean age was 39.86±7.1 years)

3.3.3. Data collection

The survey was done with the help of a pre-tested semi-structured questionnaire (See appendix 2) followed by clinical examination of the oral cavity. Besides general information's like age, occupation, education and income of the subject, information's regarding the use of smokeless tobacco, other forms of tobacco, alcohol consumption, past dental history and oral hygiene habits were asked and graded. The allotted time period was 7 weeks and a total of 66 clusters were taken for the survey.

While doing field visit in the morning, in certain houses women were alone and other households members were not there at that time. Because of these reasons some of them refused to participate in the study. Thus in the sample population the percentage of female participants is lower than in the district.

The reason for restricting the study to 30-50 age group is explained as follows.

Periodontal disease (Adult periodontitis) increases after the age of 50 because of decreased vascularity in the gingiva resulting in diminished reparative capacity concurrent with adverse effects of abundant plaque and calculus deposits. Though the consumption of tobacco products starts at an early age the adverse effects of tobacco on the periodontium starts slowly but can also raise steeply to develop periodontitis at an early stage depending on the severity of consumption. It was found that rapidly progressing periodontitis is associated with scantier amounts of plaque and calculus and

is more commonly found in the 20-35 age group and usually subsides after that age. With the consumption of tobacco products there will be an increase in plaque formation at an early age resulting in poor oral hygiene so that rapidly progressing periodontitis can change to adult periodontitis. The reason for choosing 30-50 age group is to determine whether 'adult periodontitis' is persisting in this age group for which tobacco consumption is acting as a risk factor or not.

The participants of the study were classified into four groups. Those who consume both forms of tobacco (smoking and chewing), smokers alone, chewers alone and non users of tobacco. Tobacco users included smokers and smokeless tobacco users. Direct questions were asked to the participants regarding the use of tobacco and those who were chewing or smoking or whether they had this habit previously were graded as one and those who had not used it were graded as two. A non-user is defined as those who had not used tobacco during his/her lifetime.

To assess the level of education, it was categorised into four groups-Illiterate, Primary, High school and College. Illiterates were defined as those who do not know how to read and write and had not been to school in their entire lifetime.

Those who had studied till seventh standard in schools were included in the primary level category and followed by high school level starting from eighth standard till tenth standard, followed by College level.

Occupation of the individual was graded on the basis of type of work the individual was handling and also the wage pattern of the individual. Those who were doing works involving physical activities and earning daily from that job were included in the grade one category, followed by grade two which involves people doing physical activities but getting their wages on monthly basis and office level category which also includes self employed people, were graded as three.

Socio-economic status was assessed on the basis of household income. Household income assessment was done after taking into account the inflow from all the possible sources of income in the household. Although care was taken to the large extent, it was truly difficult to desegregate the portion of the household income like the one from the household business. Even though some of the individuals were listed as 'house wives', they were productive and were generating income to the house hold to some extent. After considering all these factors, household income was categorised into low, middle and high-income groups. Low income group are those earning less than 2000 rupees per month followed by middle income group earning 2000-5000 rupees per month and more than 5000 rupees are placed in the high income group.

Oral hygiene habits of each individual was assessed on the basis of method of cleaning, frequency of cleaning and the materials used for cleaning the oral cavity.

To avoid inter observer variation, the investigator himself had examined all subjects and a dental hygienist student from Thiruvananthapuram Dental College was trained by the researcher to fill the questionnaire form.

The armamentarium for the study includes plain mouth mirrors, CPITN probe, tumblers and cidex solution for sterilisation. (Please refer appendix 4)

3.4. COMMUNITY PERIODONTAL INDEX OF TREATMENT NEEDS [CPITN INDEX]

The index that resulted after extensive field-testing by investigators from the WHO and the international Dental Federation (FDI) was called the Community Periodontal Index of Treatment Needs. The community periodontal index of treatment needs was developed by the WHO-FDI venture in 1979 and adopted in 1983.

The CPITN assesses the periodontal condition of a subject based on the following periodontal disease markers namely

0. No disease
1. Presence of gingival bleeding following probing
2. Detection of supra or sub gingival calculus
3. Measurements of shallow periodontal pockets- (4-5mm)
4. Measurement of deep periodontal pockets (6mm or more)

Specially designed CPITN probes were used for examination. CPITN probe was designed by professor George.S.Beagre of Vancouver and Professor Jukka Ainamo of Helsinki is a special periodontal probe (Ainamo et.al 1982) and has the following specifications.

1. Made of metal, handle of diameter 3.5mm, maximum height 4.5gm
2. Working tip- a sphere of 0.5mm, probe has a diameter of 0.25mm where the sphere is attached.
3. Black band- between 3.5mm and 5.5mm from the tip of the probe.

3.4.1. CPITN INDEX

The mouth was divided into sextants defined by tooth numbers 14-18,13-23,24-28,38-34,33-43and 44-48.

A sextant is examined only if there were two or more teeth present and not indicated for examination.

The third molars are not included except where they are functional in place of second molar. When only one tooth remains in a sextant it is included in adjacent sextant.

3.4.2. Index tooth

In epidemiological survey, in assessing the periodontal treatment needs of a population, the recordings per sextant are based on findings from specified index tooth. Only 10 teeth known as index teeth are examined for an epidemiological survey. The teeth have been identified as the best estimate of the worst periodontal condition of the mouth. The 10 specified index teeth are

17, 16	11	26,27
47,46	31	36,37

{17- upper right second molar, 16-upper right first molar, 11-upper right central incisor, 26-upper left first molar, 27-upper left second molar, 47-lower right second molar, 46-lower right first molar, 31-lower left central incisor, 36-lower left first molar, 37-lower left second molar}.

The molars are recorded in pairs and only one score, the highest is recorded. If in a posterior sextant if one of the molar teeth is not present then there was no replacement. If index teeth were missing then all other teeth present in that sextant were examined and

the worst score obtained is recorded. In the anterior maxillary sextant, if the tooth 11 is excluded substitute with 21, if 21 is also excluded, then identify the worst score for the remaining teeth. This substitution is also applicable to the lower teeth.

3.4.3. The probing procedure

The working force should not be more than 25grams. The gingival sulcus around each index tooth is probed in a sextant to determine pocket depth and to detect calculus and bleeding response. The probe is inserted into the periodontal pocket in such a way that the ballpoint follows the anatomic configuration of the surface of the tooth. The probe may be walked around the tooth. Each tooth is probed along four surfaces (distofacial, midfacial, mesiofacial and palatal) and only the highest score is recorded for each sextant.

3.4.4. Codes and criteria for the Community Periodontal Index Of Treatment Needs

Periodontal status	Treatment needs
0= Healthy periodontium	0=No treatment needed
1= Bleeding observed, directly or by using mouth mirror after sensing	1=Oral hygiene needs improvement
2=Calculus felt on probing but the entire black area of the probe is visible	2=1+professional scaling
3=Pocket 4 or 5mm (gingival margin is situated on the black area of the probe)	2=1+professional scaling
4=Pocket 6mm or more (black area of the probe is not visible	3=1+2+complex treatment

3.4.5. Examination procedure

Each subject was examined on an ordinary chair in adequate natural light using a mouth mirror and a probe. The examination was performed starting from the right maxillary sextant and ending at right mandibular sextant.

3.5. Data analysis

The analysis of the study was done using SPSS version 6-software program. For data entry Microsoft Excel was used. Statistical methods like Chi-Square test and multiple logistic regression analysis were used for the purpose of analysis. The analysis and entry of data were done by the investigator himself.

4. RESULTS

As stated above, 504 subjects in the age group 30-50 years from 6 panchayaths of three community developmental blocks participated in the study and the age group distribution of the sample is given below.

Table-4.1. Distribution of sample according to age group

Age group	Male	Female	Total
30-34	77(26.6%)	69(32.2%)	146
35-39	65(22.4%)	29(13.6%)	94
40-44	60(20.7%)	35(16.4%)	95
45-50	88(30.3%)	81(37.9%)	169
Total	290(100%)	214(100%)	504

Table-4.2. Based on the level of education, number of individuals in each category is shown in the table below.

Level of education	Number of individuals	Percentage
Illiterate	23	4.6
Primary	161	31.9
High school	221	43.8
College	99	19.6
Total	504	100

Of the total subjects 43.8% had been in the high school level of education followed by 31.9% in primary levels, 19.6% in college levels and 4.6% illiterates.

Table-4.3. House hold income distribution of the sample

House-hold income (rupees)	Total	Percentage
Less than 2000	282	56.0
2000-5000	114	22.6
More than 5000	108	21.4
Total	504	100

Household income wise distribution of the sample shows that 56% of the sample population were having a household income of less than 2000 rupees per month, followed by 22.6% in the 2000-5000 category and 21.4% in the higher income group category.

Table-4.4. Distribution of subjects according to occupation

Occupation	Total	Percentage
0	111	22
1	221	43.8
2	75	14.9
3	97	19.2
Total	504	100

Grade 0 - *No occupation*
Grade 1 - *Jobs involving physical activities & paid daily*
Grade 2 - *Jobs involving physical activities & paid monthly*
Grade 3 - *Office category jobs*

Of the total population, 43.8% were involved in jobs, which need physical activities and were daily wage laborers. The subjects within this category form the largest group in this study, which comprises 221 of the total sample size.

On the basis of tobacco consumption, subjects were categorised into- those using both forms of tobacco, smokers alone, chewers alone and nonusers.

Table-4.5. Distribution of subjects according to tobacco consumption

Tobacco use	Total	Percentage
Using both forms	80	15.9
Smokers alone	112	22.2
Chewers alone	65	12.9
Non users	247	49
Total	504	100

The table shows the distribution of subjects consuming tobacco in various forms and nonusers. As mentioned above, 15.9% of total sample population consumes both forms of tobacco. 22.2% of the subjects were smokers while 12.9% were chewers. Thus 51% of the subjects consume tobacco in any of the forms as mentioned above. When compared to tobacco users, 49% of the total sample population abstained from consuming any forms of tobacco.

Table-4.6. Male female comparisons of tobacco consumption

Tobacco consumption	Males	Females	Total
Using both	77(26.5%)	1(0.6%)	78
Smokers	112(38.6%)	-	112
Chewers	16(5.5%)	52(24.3%)	68
Non users	85(29.3%)	161(75.2%)	246
Total	290(100%)	214(100%)	504

The table illustrates that, the practice of using both forms of tobacco and smoking alone were predominantly common among males while in the chewer category, females were more when compared to males. In the non-user category, of the respective sample population for each sex (290 & 214 respectively for males and females) 29.3% of males and 75.2% of females were not consuming any forms of tobacco.

Table-4.7. Distribution of subjects according to alcohol consumption

Alcohol	Total	Percentage
Users	121	24%
Non users	383	76%
Total	504	100%

The use of alcohol was found to be prevalent among 24% of subjects in the total sample population. Of the 504 subjects 121 of them were having the habit of consuming alcohol.

Table-4.8. Oral hygiene habits of the sample population

Method of cleaning	Finger 131(26%)	Toothbrush 373(74%)	Others Nil
Frequency of cleaning	Once 394(78.2%)	Twice 108(21.4%)	Thrice or more 2(0.4%)
Materials used for Cleaning	Charcoal 120(23.8%)	Tooth paste 375 (74.4%)	Others 9 (1.8%)

Total sample population = 504(100%)

When taking into consideration the oral hygiene habits of the sample population, the table clearly shows that 74% of the participants uses tooth brush for cleaning the mouth followed by 26% of the subjects relying only on finger for cleaning the mouth. With regards to frequency of cleaning, 78.2% of the sample population cleans their mouth once followed by 21.4% of the population cleaning their mouth twice and only 0.4% cleaning their mouth thrice. When considering the materials used for cleaning, it was found that 74.4% of the participants used toothpaste for cleaning, followed by charcoal 23.8% and others 1.8%.

Table-4.9. Materials and methods used for cleaning

Materials used for cleaning	Method of cleaning		
	Finger	Tooth brush	Total
Charcoal	113(94.2%)	7(5.8%)	120(23.8%)
Tooth paste	18(4.8%)	357(95.2%)	375(74.4%)
Others		9	9(1.8%)
Total	131(26%)	373(74%)	504

While correlating the materials and methods used for cleaning, it was found that, of the 120 subjects using charcoal, 94.2% of them were relying on their finger for cleaning the mouth and out of the 375 subjects using tooth paste 95.2% had been found using tooth brush for cleaning.

As stated above one of the main objectives of the study was to determine the prevalence of periodontal disease in a sample population of Thiruvananthapuram district.

A patient can be diagnosed as having periodontal disease if he/she is having a true periodontal pocket, which is a pathologically deepened gingival sulcus. In this study, the

'Community periodontal Index of treatment needs' was used to assess the periodontal condition and treatment needs of the study sample. According to the criteria set for the index, those subjects falling under the grade 3 category (having a pocket depth of 4 - 5mm) and grade 4 category (indicating a pocket depth of more than 5.5mm) were diagnosed as having the disease.

Table-4.10. Periodontal disease prevalence among the study population

Disease present	329	65.3%
Disease absent	175	34.7%
Total	504	100%

From this table, according to Community Periodontal Index of Treatment Needs it is clear that, 329(65.3%) of the 504-sample population were affected with periodontal disease (95%CI 61.1-69.4). Among the 329 subjects 91(27.7%) of them were having deep periodontal pockets (CPITN4) and 238 (72.3%) were having shallow pockets (CPITN3).

Table.4.11. Periodontal disease prevalence in different panchayaths

Name of the panchayath	Disease present	Disease absent	Total
Vellanad	52(62.7%)	31(37.3%)	83(100%)
Poovachal	54(69.2%)	24(30.8%)	78(100%)
Chirayankeezhu	65(60.2%)	43(39.8%)	108(100%)
Kizhuvilam	60(69%)	27(31%)	87(100%)
Vamanapuram	59(81.9%)	13(18.1%)	72(100%)
Pulimath	39(51.3%)	37(48.7%)	76(100%)
Total	329	175	504

The table 4.11 illustrates that among the 6 panchayaths selected for the study, prevalence of the disease varied from 51.3% in Pulimath panchayath to 81.9% in Vamanapuram panchayath.

Table-4.12. Education and periodontal disease

Level of education	Disease present	Disease absent	Total
Illiterates	18(78.3%)	5(21.7%)	23(100%)
Primary	123(76.4%)	38(23.6%)	161(100%)
High school	145(65.6%)	76(34.4%)	221(100%)
College	43(43.4%)	56(56.6%)	99(19.6%)
Total	329	175	504

Among the total sample population, 221 of them had studied till the high school level (largest group in the study sample) Among that group 65.6% were affected by the disease. The worst affected groups were illiterates (78.3%) and those belonging to the primary school level category (76.4%). The decrease of periodontal disease with highest level of education may be due to awareness among the people regarding the disease and access to dental health care facilities within their reach. Chi-square test had shown a statistically significant association between education and periodontal disease ($p<0.001$).

Table-4.13. Occupation and periodontal disease

Occupation	Disease present	Disease absent	Total
Grade zero	75(67.6%)	36(32.4%)	111(100%)
Grade one	166(75.1%)	55(24.9%)	221(100%)
Grade two	39(52%)	36(48%)	75(100%)
Grade three	49(50.5%)	48(49.5%)	97(100%)
Total	329	175	504

Grade zero- no occupation
Grade 1 - Jobs involving physical activities & paid daily
Grade 2 - Jobs involving physical activities & paid monthly
Grade 3 - Office category jobs

Occupation wise distribution of the sample reveals that those who were involved in jobs involving physical activities forms the largest group among the study population. 75% of the subjects in this group were affected with disease. It may be due to the fact that, because of the type of job in which they were involved they were consuming tobacco much more than others which can be one reason for increase in disease prevalence in daily wage workers.

Table-4.14. House hold income (in rupees) and periodontal disease

Household Income (Rs.)	Disease present	Disease absent	Total
< 2000	210(74.5%)	72(25.5%)	282(100%)
2000-5000	61(53.5%)	53(46.5%)	114(100%)
>5000	58(53.7%)	50(46.3%)	108(100%)
Total	329	175	504

The disease prevalence was very high among the first group (<2000 rupees) when compared to other groups. In rest of the group's disease prevalence is almost similar.

The reason for decrease in periodontal disease with increase in income may be due to the paying capacity of the subjects for treatment procedures, which were found unaffordable to those in the lowest socio-economic status. The Chi-square test had shown a statistically significant association between household income and periodontal disease ($p<0.001$).

Table-4.15. Male female comparisons of periodontal disease of the population

Sex	Disease present	Disease absent
Male	203(61.7%)	87(50%)
Female	126(38.2%)	88(50%)
Total	329(100%)	175(100%)

The table 4.15 points on to the male female comparisons of periodontal disease of the study population. There is a clear difference in the prevalence of periodontal disease between men and women. The prevalence of periodontal disease among males was 61.7%(95%CI 56.11, 67.29) and that among females was 38.2%(95%CI 31.7,44.7). Chi-square test shows that this association is also statistically significant. ($p<0.001$).

Table-4.16. Periodontal disease status of the population in relation to different age groups

Age group	Disease present	Disease absent	Total
30-34	66(45.2%)	80(54.8%)	146(100%)
35-39	57(60.6%)	37(39.4%)	94(100%)
40-44	70(73.7%)	25(26.3%)	95(100%)
45-50	136(80.5%)	33(19.5%)	169(100%)
Total	329	175	504

The maximum disease prevalence is seen in the 45-50 age group. Of the 169 subjects in this age group, 80.5% were having periodontal disease. This table reveals the prevalence of periodontal disease in different age groups and clearly shows that as age increases periodontal disease also increases. Chi-square test shows positive association between age group and the periodontal disease (p value <0.001).

Table- 4.17. Periodontal disease status among tobacco users and non users

Tobacco Use	Disease present	Disease absent	Total
Using both	62(79.5%)	16(20.5%)	78(100%)
Smokers alone	88(78.6%)	24(21.4%)	112(100%)
Chewers alone	44(64.7%)	24(35.3%)	68(100%)
Non users	135(54.9%)	111(45.1%)	246(100%)
Total	329	175	504

The table 4.17 had categorized subjects into four groups. Those who were consuming both forms of tobacco, smokers alone, chewers alone and non-users of tobacco. The table points to the fact that 80% of the subjects consuming both forms of tobacco were prone to disease. While comparing both smokers and chewers it was observed that 79% of smokers alone were having the disease where as only 64.6% of chewers were affected with disease. In the non-user category, of the total 246 subjects 55% were having disease. Here again Chi-square test had shown a statistically significant association (p<0.001).

Table-4.18. Alcohol and periodontal disease

Alcohol Consumption	Disease present	Disease absent	Total
Users	95(78.5%)	26(21.5%)	121(100%)
Non users	234(61%)	149(39%)	383(100%)
Total	329	175	504

As it can be seen from the table, 78.5% of alcohol users were having the disease when compared to nonusers, which constitutes only 61% of that group. The chi-square test had shown a statistically significant association between alcohol and periodontal disease ($p<0.001$).

As stated above the oral hygiene habits of the study population is classified on the basis of method of cleaning, frequency of cleaning and materials used for cleaning the oral cavity.

Table-4.19. Periodontal disease status in relation to method of cleaning

Method of cleaning	Disease present	Disease absent	Total
Finger	106(80.9%)	25(19.1%)	131(100%)
Tooth brush	223(59.8%)	150(40.2%)	373(100%)

While taking into consideration the method of cleaning the oral cavity, among the study population, it was found that of the 131 participants, who were using only their finger for cleaning, 81% of them were having periodontal disease and of the 373 participants using

toothbrush, 60% were affected with periodontal disease. Chi-square test had shown a statistically significant association between method of cleaning and periodontal disease (p value<0.001).

Table-4.20. Periodontal disease status in relation to frequency of cleaning

Frequency of cleaning	Disease present	Disease absent	Total
Once	273(69.3%)	121(30.7%)	394(100%)
Twice	56(50.9%)	54(49.1%)	110(100%)
Total	329	175	504

From the table 4.20 shown above, it is clear that of the 394 sample population who were having the habit of cleaning their mouth once, 69.3% of them were affected by disease, where as among those who cleans their mouth twice (110), only 51% were affected. Chi-square test reveals a statistically significant association between frequency of cleaning and periodontal disease (p value=0.003).

Table-4.21. Periodontal disease status of the study population in relation to materials used for cleaning

Materials used	Disease present	Disease absent	Total
Charcoal	98(81.7%)	22(18.3%)	120(100%)
Tooth paste	225(60.%)	150(40%)	375(100%)
Others	6(66.7%)	3(33.3%)	9(100%)
Total	329	175	504

Table 4.21 shows that, of the 120 sample population using only charcoal for cleaning, 81.7% of them were having the disease, while among the 375 sample population using

tooth paste alone 60% were affected by periodontal disease. Here again the Chi-square test had shown a statistically significant association between materials used for cleaning and periodontal disease ($p<0.001$).

While correlating materials and methods used for cleaning it was found that of the 375 subjects using toothpaste, 357 of them use tooth brush (total tooth brush users were 373) while rest of them rely on fingers. In the case of charcoal users 113 of the total 120 users rely on finger and 7 of them use toothbrush.

Table-4.22.Prevalence of periodontal disease in relation to oral hygiene habits (Combination of methods and materials used for cleaning)

Oral Hygiene Habits	Disease present	Disease absent	Total
Finger & charcoal	95(84%)	18(16%)	113(100%)
Finger & paste	11(61%)	7(39%)	18(100%)
Brush & charcoal	3(43%)	4(57%)	7(100%)
Brush & paste	214(60%)	143(40%)	357(100%)
Brush & others (salt & tooth powder)	6(67%)	3(33%)	9(100%)
Total	329	175	504(100)

The above table illustrates the prevalence of periodontal disease in relation to oral hygiene habits. When combining materials and methods used for cleaning and relating with periodontal disease it was found that the disease prevalence was more prevalent among finger and charcoal users constituting 84% within that group.

From the above bivariate comparisons to find out the association between periodontal diseases and selected variables, the statistically significant selected variables were age group, education, socioeconomic status (household income), oral hygiene habits like frequency of cleaning, materials for cleaning, methods for cleaning, sex, tobacco users and alcohol.

Multivariate analysis using multiple logistic regression was attempted to study the associations adjusted for several variables simultaneously. The table below represents

the logistic regression results of the dependent variable periodontal diseases with selected independent variables taken for the study. The independent variables selected were age group, education, Socio-economic status (household income), oral hygiene habits like frequency of cleaning, materials for cleaning, methods for cleaning, sex, tobacco users and alcohol.

Results of the multivariate logistic regression: Dependent variable: Periodontal disease. Independent variables were age group, education, Income, frequency of cleaning, materials used for cleaning, methods of cleaning, sex, tobacco and alcohol

The table is given in the succeeding page

Table-4.23.

Dependent variable- periodontal disease			
Independent variables	**Odds ratio**	**p-value**	**95%CI**
Age group			
30-34	1		
35-39	1.71	.07	(0.94,3.10)
40-44	2.96	<. 001	(1.58,5.54)
45-50	5.48	<. 001	(3.01,9.96)
Education			
Illiterates	1		
Primary	1.12	0.843	(0.35,3.55)
High school	1.27	0.697	(0.36,4.37)
College	0.86	0.831	(0.21,3.46)
Income(rupees)			
2000-5000	1		
<2000	0.54	0.042	(1.01,3.37)
>5000	0.55	0.947	(0.52,1.99)
Oral hygiene			
Frequency of cleaning (1)	1		
Frequency of cleaning (2)	0.69	0.166	(0.41,1.17)
Materials for-cleaning			
Charcoal	1		
Tooth paste	0.87	0.804	(0.30,2.55)
Others	0.98	0.988	(0.15,6.28)
Methods of -cleaning			
Finger	1		
Tooth brush	0.69	0.482	(0.24,1.96)
Sex			
Males	1		
Females	1.44	0.238	(0.77,2.67)
Tobacco			
Non users	1		
Using both	1.90	0.147	(0.78,4.61)
Smokers	2.65	0.006	(1.29,5.41)
Chewers	1.07	0.917	(0.28,4.07)
Alcohol			
Users	1		
Nonusers	0.54	0.076	(0.27,1.08)

The table 4.23 shows the results of multivariate analysis. (Interaction term sex * tobacco were also included, but was not statistically significant)

The variables that were significant in bivariate analysis lost significance in multivariate analysis.

As mentioned above, the independent variables which had shown statistically significant associations were age group, sex education, household income, tobacco, oral hygiene habits like method of cleaning, frequency of cleaning, materials used for cleaning and alcohol. After doing multivariate analysis it was found that the variables, which showed statistically significant association with periodontal disease, were age group, socio-economic status and tobacco consumption. With reference to age, the table clearly illustrates that with increase in age the prevalence of disease had also increased and was found maximum in the 45-50 age group ($p<0.001$). The odds ratio for this group was found to be 5.5. This means that the subjects with in this age group had 5.5 times odds of having periodontal diseases when compared to other groups. With reference to socio-economic status the table points to the fact that, the disease prevalence is more among subjects having an income of less than 5000 rupees (p value=0.042). With an increase in income (>5000 rupees) the p value had lost its significance.

A statistically significant association had been found between cigarette/bidi smoking and periodontal disease. Logistic regression had shown that when compared to the rest of the groups smoking has became a major risk factor responsible for developing the disease (p value=0.006 and odds ratio=2.6). From the results its clear that, smokers had 2.6 times odds of developing disease when compared to those using both forms of tobacco, chewers and non users.

5. DISCUSSION AND CONCLUSIONS

It is important in the middle years of life and later to continue good oral hygiene habits and the practice of having regular dental checkups. It was found that periodontal disease increases in the middle years of life, which is the major reason for teeth loss in adults. Tobacco consumption has got substantial influence on the periodontal health and disease of a society. Smoking is one of the most important risk factor for periodontal disease. Even though the pathogenesis of periodontitis in smokers is incompletely understood, epidemiological data indicate a direct effect. When compared to smokeless tobacco, the most consistently reported periodontal effects of smokeless tobacco are gingival recession and mucosal lesions; some forms of periodontal diseases are also reported. Though the association between smoking and periodontal disease is widely accepted, the association between smokeless tobacco and periodontal disease is still a debatable issue.

Past epidemiological surveys held in India indicate a very high prevalence of periodontal diseases with varying degrees of severity[35,36,37]. Very few epidemiological studies have been reported from Kerala[9,37]. Gupta surveyed the general population of Trivandrum aged 11-80 years and found 100% prevalence for gingivitis from age 31 onwards.

The overall picture of periodontal diseases that emerged from the CPITN data collected from 6 panchayaths in Thiruvananthapuram district resembled the CPITN data obtained from other parts of the world. The gradual worsening of the periodontal conditions with increasing age reflected the chronic progressive nature of periodontal disease.

The present study was conducted to estimate the prevalence of periodontal disease among the population and also to assess the prevalence of periodontal disease associated with tobacco consumption in the community.

In this study the index used for assessing the periodontal status of the population was Community Periodontal Index Of Treatment Needs (CPITN). The CPITN is an ideal index for epidemiological studies because it uses accepted clinical criteria, partial mouth scoring and a simple recording procedure, which allows rapid assessment of individuals for periodontal conditions related to treatment needs.

This study was conducted in 6 panchayaths of three community developmental blocks in Thiruvananthapuram district. 504 people in the age group 30-50 participated in the study. In our study, 329(65.3%) of the subjects were found to have periodontal disease.

From the bivariate comparisons used to find out the association between periodontal diseases and selected variables, the statistically significant selected variables were age group, education, socioeconomic status (household income), oral hygiene habits like

frequency of cleaning, materials for cleaning, methods for cleaning, sex, tobacco users and alcohol.

5.1. Tobacco and disease

The participants in our study according to tobacco consumption were broadly classified into four groups-those who were using both forms of tobacco, smokers alone, chewers alone and non-users of tobacco. It was found that the disease prevalence was more in those who were consuming both forms of tobacco (79.5%), followed closely by smokers (78.6%). From the above information it is clear that smokers and those who were using both forms of tobacco were affected more by the disease. In chewers and non-users of tobacco, the percentage was found to be lower when compared to first two categories (64.7%and 54.9% respectively). Multivariate analysis using multiple logistic regressions was attempted to study the associations adjusted for several variables simultaneously. The variables, which were significant in bivariate analysis, lost significance in multivariate analysis. From multivariate analysis it was found that smoking, household income and age group were associated with periodontal disease. In the case of tobacco consumption, when adjusted for multivariate analysis it showed that smoking alone was the sole factor responsible for periodontal disease (P=0.006). It should also be noted that in subjects who were consuming both forms of tobacco, the association was not statistically significant. It may be due to the fact that, tobacco chewing may have masked the harmful effects of smoking and this factor had lead to an insignificant association. The odds ratio for smoking alone was found to be 2.6. From the results its clear that smokers had 2.6 times odds of developing disease when compared to those using both,

chewers and non users. It may be due to the fact that, smokers have poor oral hygiene, which leads to an increase in plaque and calculus formation, and many studies had supported this fact[17,18]. There is a growing body of scientific evidence indicating that nicotine contributes to the progression of periodontal disease. Exposing periodontal tissues to tobacco smoke may effect periodontal break down in two potential pathways: tobacco smoke may function directly as a source of gingival irritation; and indirectly a systemic mechanism may alter vascular tissue and haemodynamics.[39] As a result of gingival vasoconstriction decreased number of immune cells are available in the gingival tissue, which translates into a weakened defence-reparative system. Many studies have shown that smoking is the main factor responsible for periodontitis[4,5,38].

When considering tobacco chewing alone it was found that this factor alone is not responsible for periodontal disease formation. Robertson P.B et al (1990)[40] in their study "Periodontal effects associated with the use of smokeless tobacco" in professional foot ball players reported that 46% had oral mucosal lesions, located primarily in the mandible at sites where the smokeless tobacco quid was placed. They also reported that the use of smokeless tobacco was not necessarily associated with severe forms of periodontal disease though sites adjacent to mucosal lesions in smokeless tobacco users showed significantly greater recession and attachment loss than in sites not adjacent to lesions in users or comparable sites in non-users. Similar findings have been observed by Thomas and Johnson (1987)[41] in their study "Smokeless tobacco and its effect on oral tissues". They reported that tobacco chewers show greater destruction with respect to gingival recession whereas chewing of tobacco did not cause any other destructive changes in the

periodontium. The exact reason for decrease in periodontitis in tobacco chewers is unknown. One of the arguments is that, while chewing the betel quid there will be an increase in salivary flow. The flow causes a physical cleaning action and a biochemical action and due to these reasons the bicarbonate content, pH and buffering capacity are all greatly increased which has its effect on the oral cavity.[31]

In the bivariate analysis one of the variables having significant association with periodontal disease was sex. In this analysis males were found to have disease more when compared to females and it can be due to the fact that smoking prevalence was more among males. The Chi-square test had shown a statistically significant association between sex and disease. After doing multivariate analysis when adjusted for other variables, it was found that the variable sex had lost its significance.

5.2. Age group and periodontal disease

While taking into consideration the periodontal disease status among different age groups, it was found that with advancing age shallow and deep periodontal pockets (grades 3&4) had remarkably increased and were highest in the 45-50 year age group (81%). This gradual worsening of periodontal conditions with increasing age reflects the progressive nature of periodontal disease. The progressive nature of the disease with increasing age was also reported by Anil S et al (1990)[9] in their study conducted to find the periodontal conditions of a selected population in Trivandrum district. In their study, the disease prevalence in the 35-44 age group was very high. The study reports that 77% of the subjects belonging to that particular age group were having the disease and when compared to the younger age group, this was found to be very high.

5.3.Socioeconomic status and periodontal disease

The disease prevalence was very high in the poorest socioeconomic group when compared to other groups. In rest of the group's disease prevalence was almost similar. The reason for decrease in periodontal disease with increase in income may be due to the paying capacity of the subjects for treatment procedures, which were found unaffordable to those in the lowest socio-economic status. The Chi-square test had shown a statistically significant association between income and periodontal disease ($p<0.001$).

5.4.Treatment needs of the population

When considering the treatment needs of the population it should be noted that healthy periodontium was not observed in any of the subjects. Though we have taken into account CPITN 3 and CPITN 4 representing shallow and deep periodontal pockets representing periodontal disease in our study which constitutes 329 subjects (65.3%) of the entire sample population, the rest of the sample population were having calculus formation or CPITN 2 which was not taken into account because we were considering only the disease factor. It should also be noted that among the 329 subjects 91 of them were having deep periodontal pockets and for this section of subjects the treatment procedures include oral hygiene instructions, complete scaling followed by advanced treatment procedures, which includes surgery. The rest of the subjects needed oral hygiene instructions and scaling. (The treatment for CPITN 2 and CPITN 3 includes oral hygiene instructions and scaling only. In some cases with shallow pockets, curettage is also necessary) In general, although both smoking and chewing has a detrimental effect

on the periodontium, the severity of periodontal destruction with respect to periodontal pocketing was more in smokers than in chewers.

5.5.Limitations

Contrary to the population census in Kerala where females are more compared to males, the male female ratios in this study is favoring male population.

The instrument CPITN probe used for the study was calibrated so that no second attempt was taken to check the validity of the results.

5.6.Conclusions

In our community based study, around two thirds of the adult population had periodontal disease. The prevalence of disease was also more in older age groups when compared to younger age groups indicating a possible progression of disease with advancing age. The emergence of smoking as a risk factor for periodontal disease should be dealt with utmost importance. The results that emerged from this epidemiological study also suggest that the socioeconomic status of the population has a direct bearing on the prevalence of periodontal disease. Educational efforts targeted to reduce smoking should be considered at the community level. Dental checkups should be started in an early age considering the progressive nature of the disease with age.

5.7.Policy implications

The study highlights on the fact that this poor state of periodontal health may be related to lack of proper dental health knowledge, expensive treatment procedures and the virtual

absence of preventive and therapeutic dental services. While taking into consideration the prevalence rate of the disease among the population, educational efforts at national and local levels are needed to focus both public and professional attention on the problem of periodontal diseases and its association with tobacco consumption. An obvious implication is that smoking prevention should also be included in dental public health education programme for the purpose of preventing periodontal disease. Furthermore highest priority should be assigned to educating children and youth not to start tobacco use.

Dental treatment procedures are inherently expensive and as a result of this, many communities are not able to afford even essential treatment procedures. Compared to the prohibitive cost of dental care provided by private institutions, government run dental colleges and district hospitals provide relatively inexpensive care to the public. However, only a small fraction of the population is able to avail of this facility.

In order to overcome this problem, the provision of dental care should be extended to primary health centres and also made accessible to people living in rural areas. Dental hygienists can also play a major role in controlling the early onset of periodontal disease. They should be acquainted with field visits and trained in conducting epidemiological surveys. This will help them to detect the onset of the disease in its early stages. Thus, they can provide treatment to patients who are in the preliminary stages of infection and also refer advanced cases to the concerned specialist.

6. REFERENCES

1. The World Health Report 1997. "Conquering suffering and enriching humanity". (Report of the Director General), World Health Organization Geneva, 1997; 71.
2. Semmelweis Orvostudomanyi Egyetem. "The effect of smoking on the spread and frequency of periodontal disease". Forgov SZ, 1999; Apr;92(4):99-110.
3. QuandilR, Sandhu HS, Mathews DC. "Tobacco smoking and periodontal diseases". Journal of Canadian Dental Association 1997, March; 63(3): 187-92, 194-5.
4. Turnbull B. "Smoking and periodontal disease". JNZ Soc Periodontol, 1995;79: 10-5.
5. Semmelweis Orvostudomanyi Egyetem. "The effect of smoking on the spread and frequency of periodontal disease". Forgov SZ, 1999; Apr;92(4):99-110.
6. Kerr NW. "Prevalence and natural history of periodontal diseases in Scotland-the mediveal period"(900-1600 AD). Journal of Perio Res, 1991; 26: 346.
7. Fermin A.Carranza,jr,Dr.Odont, Glickmans clinical periodontology. (7th edition)1990; W.B. Saunders, Philadelphia:400.
8. ChristenAG. "Clinical effects of tobacco use on oral tissues". JADA,1970;81:1379.
9. Anil S, Hari S, Vijayakumar T. "Periodontal conditions of a selected population in Trivandrum district, Kerala, India". Community dental oral epidemiology, 1990;18:325.
10. Preber H, Bergstrom J, "Cigarette smoking in patients referred for periodontal treatment". Scand J Dent Res, 1986; 94: 102-108.
11. Tonetti MS. "Cigarette smoking and periodontal diseases:etiology and management of disease". Ann periodontol, 1998;1:88-101.

12. Rajala.M, Honkala E, Rimpela M and Lammi. "Tooth brushing in relation to other habits in Finland". Community Dentistry and Oral Epidemiology, 1980;8:391-395.

13. Taani DS. "Association between cigarette smoking and periodontal health". Quintessence Int, 1997; 28: 535-9.

14. Weinstein RL, Francetti L, MaggioreE, MarchesiG. "Alcohol and smoking. The risk factors for the oral cavity". Minerva Stomatol, 1996; 45:405-13.

15. Sachit Anand Arora. "Comparative evaluation of periodontal status between cigarette smokers and tobacco chewers using PSR index". Unpublished study.

16. Jette AM, Feldman HA, Tennstedt Sl . "Tobacco use: a modifiable risk factor for dental disease among the elderly".American journal of public health,1993; 83:1271-6.

17. Arno A, Schei O, LovdalA and Wearhaug J . " Incidence of gingivitis as related to sex, occupation, tobacco consumption, tooth brushing and age". Oral Surg, 1958; 11: 587-595.

18. Bergstrom j, Eliasson, PreberH. Cigarette smoking and periodontal bone loss. Journal of periodontology, 1991; 62: 242-246.

19. Schenkein HA, Gunsolley JC, Koertge TE, Tew JG. "Smoking and its effects on early onset periodontitis". JADA, 1995; 126: 1107-1113.

20. Haber J, Kent RL. "Cigarette smoking in a periodontal practice".Journal of periodontology,1992; 63:100-106.

21. Bergstrom J, Floderus-Myrhed B. "Co-twin control study of the relation ship between smoking and some periodontal disease factors". Community Dental Oral Epidemiology, 1983;11:113-116.

22. Bergstrom J, Eliasson S. "Cigarette smoking and alveolar bone height in subjects with

high standards of oral hygiene". Journal of Clinical Periodontology, 1987;14:466-469.

23. Bergstrom J, Eliasson S. "Noxious effect of cigarette smoking on periodontal health". Journal of Periodont Res, 1987; 22: 513-517

24. R.B.Bhonsle, P.R Murti, P.C.Gupta (1992). "Tobacco habits in India".In Praksh.C.Gupta, James E Hamner and P.R.Murti, Basic Dental Research Unit and WHO collaborating centre for oral cancer prevention(79-82). Tata Institute of Fundemental Research, Bombay, India. Control of tobacco related cancers and other diseases. Proceedings of an international symposium TIFR, Bombay.

25. GreerRO, Poulson TC, Boone ME. "Smokeless tobacco associated oral changes in juvenile and geriatric patients: Clinical and histopathological features". Gerodontics, 1986; 2:87.

26. Christen A.J. "The clinical effects of tobacco on oral tissues". JADA, 1970; 81:1378-1382.

27. Greer RO Jr,Poulson TC. " Oral tissue alterations associated with the use of smokeless tobacco by teen agers". J Clinical findings, Oralsurgery, 1983; 56: 275-84.

28. William H.Bowles, Mitchel R Wilkinson, Martin J.Wagner. "Abrasive particles in tobacco products". JADA,1995;126: 327-331.

29. Lindemeyer RG, Baum RH, Hsu SC. "Invitro effects of tobacco on the growth of oral cariogenic streptococci". JADA, 1981;103:719-22.

30. Offenbacher,Weathers D.R. "Effects of smokeless tobacco on the periodontal, mucosal and caries status of adoloscent males". Journal of Oral Pathology, 1985;14: 169-181.

31. ShanonT.T, Trodahl J.N. "Sugars and fluoride in chewing tobacco and snuff". Text

Dent Journal, 1978; 96: 6-9.

32. Arden G.Christen, Wilson R.Armstrong, Raymond K.McDaniel. "Intraoral leukoplakia, abrasion, periodontal break down and tooth loss in a snuff dipper". JADA,1979;98: 584-586.
33. Van Wyk, CW. "The oral lesion caused by snuff. A clinico-pathological study". J Dent Assoc S. Africa,1966; 21:109-116.
34. Pathak.J.N, C.P.Boghani. "The role of tobacco chewing in periodontal destruction". JIDA, 1979; 51: 170-176.
35. Green J.C. "Periodontal disease in India- Report of an epidemiological study" J.Dent.Res, 1960, 39: 302.
36. Chawla T.N, Nanda R.S, Mathur M.N. "Prevalence of periodontal disease in urban lucknow(India) using Ramfjord's technique". J. All Ind. Dent. Asso, 1963; 35: 151.
37. Gupta OM P." Epidemiological study of periodontal diseases in Trivandrum" J. Dent. Res, 1964;43: 876.
38. Jerome Haber, Julianne Wattles, Maureen Crowley, Robert Mandell, Kaumudi Joshipura, Ralph L Kent."Evidence for cigarette smoking as a major risk factor for periodontitis". Journal of periodontology, 1993; 64:16-23.
39. Summers CJ, Oberman A. "Association of oral disease with 12 selected variables". Journal of Dental Research, 1968; 47:457.

40. Robertson PB, Walsh M, Greene J, Ernster V, Grady D, Hauck W. "Periodontal effects associated with the use of smokeless tobacco". Journal of periodontology, 1990; 61: 438-43.

41. Thomas K, Johnson H. "Smokeless tobacco and its effect on oral tissues". Adv Dent Res, 1997;2:34-38.

APPENDIX-1

The Periodontium

The periodontium is a connective tissue organ, covered by epithelium that attaches the teeth to the bones of the jaws and provides a continually adapting apparatus for support of teeth during function. The periodontium comprises of four connective tissues. They are the cementum, alveolar bone, periodontal ligament and the gingiva. The gingiva is a part of the oral mucosa that covers the alveolar process of the jaws and surrounds the neck of the tooth. The periodontal ligament is the connective tissue structure that surrounds the root and connects it to the bone. The cementum is a calcified tissue that forms the outer covering of the anatomical root of the teeth. The alveolar process is the bone that forms and supports the tooth sockets.

The course of periodontal disease

Many types of deposits exist on the tooth surface above and below the gingival margin. The term plaque is used universally to describe the bacteria associated with tooth surface and calculus is the attached dental plaque that has undergone mineralization. The combination of bacterial action and the roughness of the resulting calculus injures the

surrounding gum tissues and makes it susceptible to infection and recession. The irritation causes swelling, inflammation and bleeding into the crevices between the teeth and gums, which is one of the early signs of impaired tissue health. The inflammation of the gums known as gingivitis can spread to the roots of the teeth if not treated. The gums separate from the teeth, forming pockets that fill up with more food particles and colonies of bacteria. As the disease progress, the bone support for the teeth is weakened and the affected teeth begin to loosen and drift from their normal position. Finally unless the disease is treated in time, the teeth may be lost.

Diagnosing a patient with periodontal disease

A patient can be diagnosed as periodontal disease if he/she is having a true periodontal pocket, which is a pathologically deepened gingival sulcus. This is the most important clinical feature of periodontal disease. The only reliable method of locating periodontal pockets and determining their extent is careful probing of the gingival margin along each tooth surface. The clinical signs that suggest the presence of true periodontal pockets are

1. *Enlarged bluish red marginal marginal gingiva with a 'rolled edge' separated from tooth surface.*
2. *A break in the facio-lingual continuity of the interdental gingiva.*
3. *Shiny, discolored and puffy gingiva associated with exposed root surface.*
4. *A reddish blue vertical zone extending from the gingival margin to the attached gingiva and some times to the alveolar mucosa.*

{The terminology's used here were taken from Glickman's Clinical periodontology seventh edition 1990}

APPENDIX-2

QUESTIONNAIRE

1. Date of Interview:

2. Panchayath name and number:

3. Ward name and number:

4. House name/ number:

5. Total residents in the household:

6. Number of earning members in the household:

7. Name of the respondent:

8. Age:

9. Sex: (1.Male 2. Female)

10. Address:

11. Education status: (1.Illiterates 2. Primary level 3. High school level 4.College level)

12. Occupation: (1.Daily wageworkers 2.Monthly wage workers 3.Office category)

13. House hold Income in rupees (1.<2000 2. 2000-5000 3. >5000)

    1. Low 2. Middle 3. High

14. Past Dental history:

    Did you visit a dentist for any oral disease (1. Yes 2. No)

15. Do/ did you chew tobacco: (1. Yes 2. No 3. Occ 4. Past)

16. If yes why you started chewing: (1. To relieve toothache 2. Other reasons)

17. How many times do you chew per day: (1. Once 2. Twice 3. Thrice 4. More)

18. How long have you been chewing:

19. Are you aware of any health hazards due to tobacco consumption: (1. Yes 2. No)

20. Do you keep the quid in the mouth while asleep: (1. Yes 2. No)

21. Do/ did you smoke: (1. Yes 2. No)

22. What do you smoke: (1. Bidi 2. Cigarette 3. Both 4. Others)

23. How many times do you smoke per day: (1. Once 2. Twice 3. Thrice 4. More)

24. Do you take alcoholic drinks: (1. Yes 2. No)

25. If yes for how long:

26. Oral hygiene habits:

    a. Method of cleaning- 1. Finger 2. Tooth brush 3. Others

    b. Frequency of cleaning- (1. Once 2. Twice 3. Thrice 4. More)

    c. Materials used- 1. Charcoal 2. Salt 3. Tooth powder 4. Tooth paste 5.Others

Periodontal Index

CPITN INDEX (Community periodontal Index Of Treatment Needs)

Community Periodontal Index

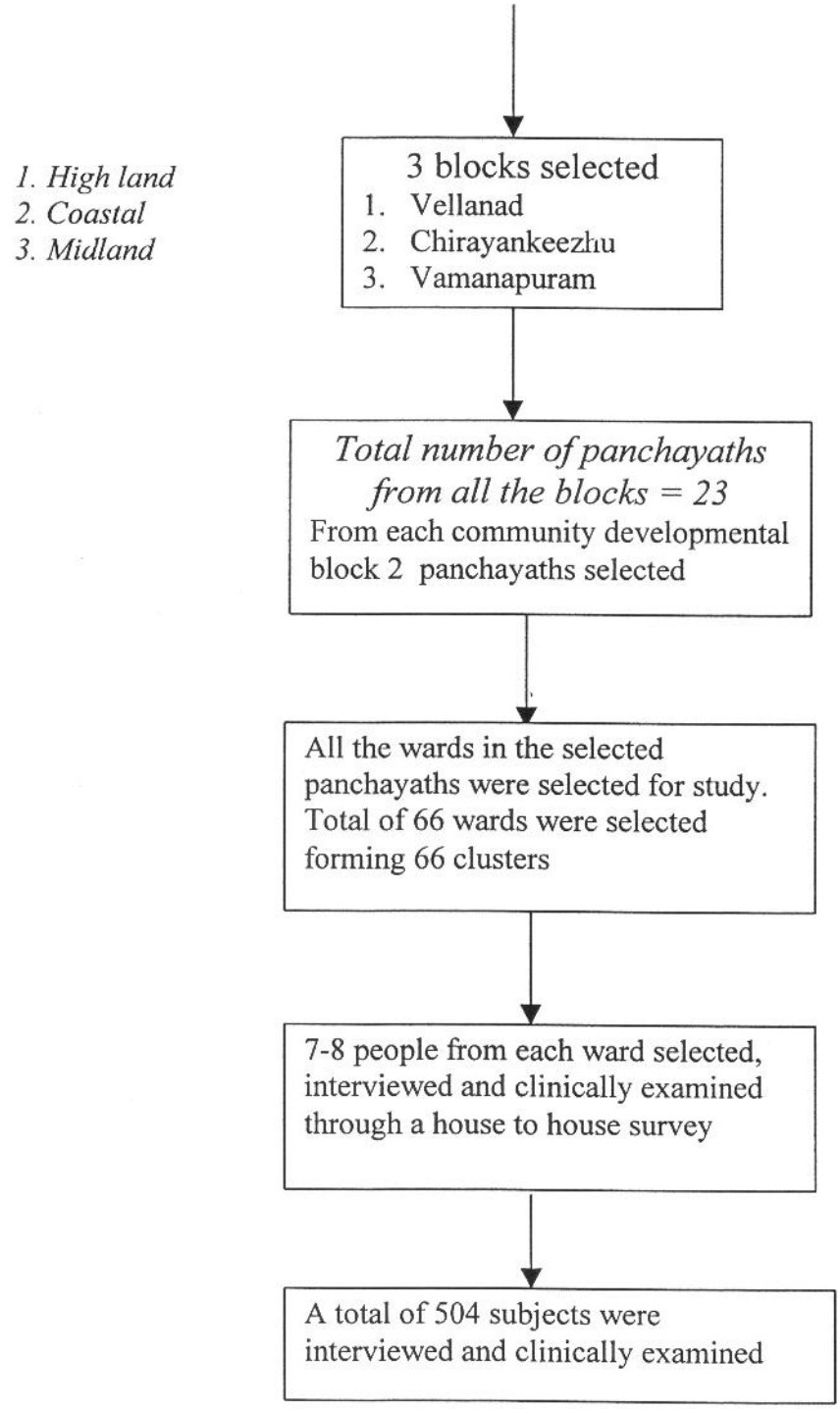
Treatment Needs

Appendix 3

SCHEMATIC REPRESENTATION OF THE STUDY CONDUCTED IN THIRUVANANTHAPURAM DISTRICT

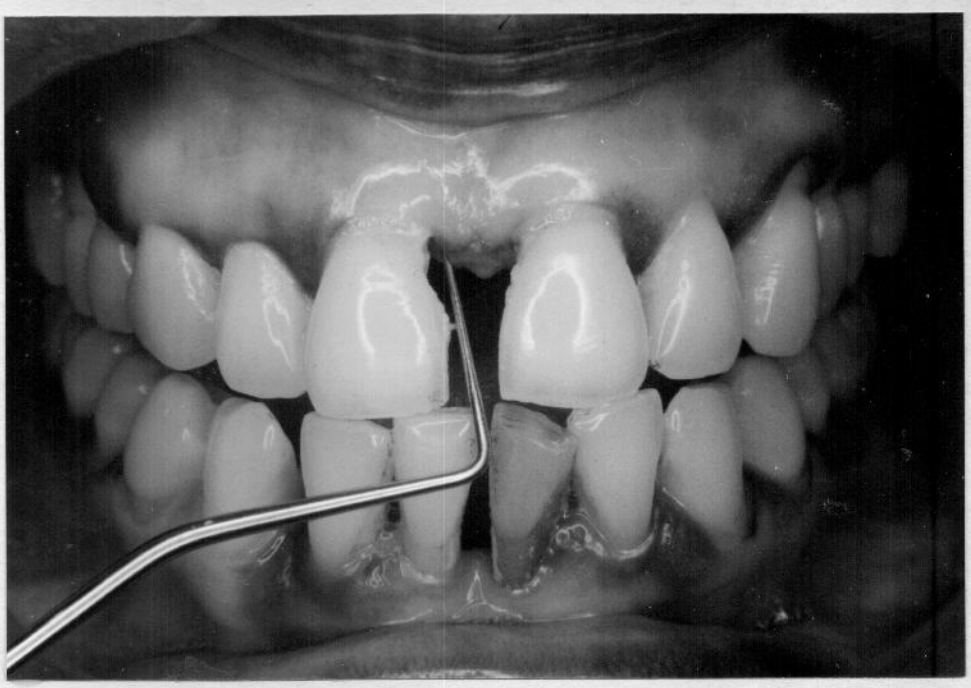
Thiruvananthapuram district (Rural)

Total Community developmental blocks = 11

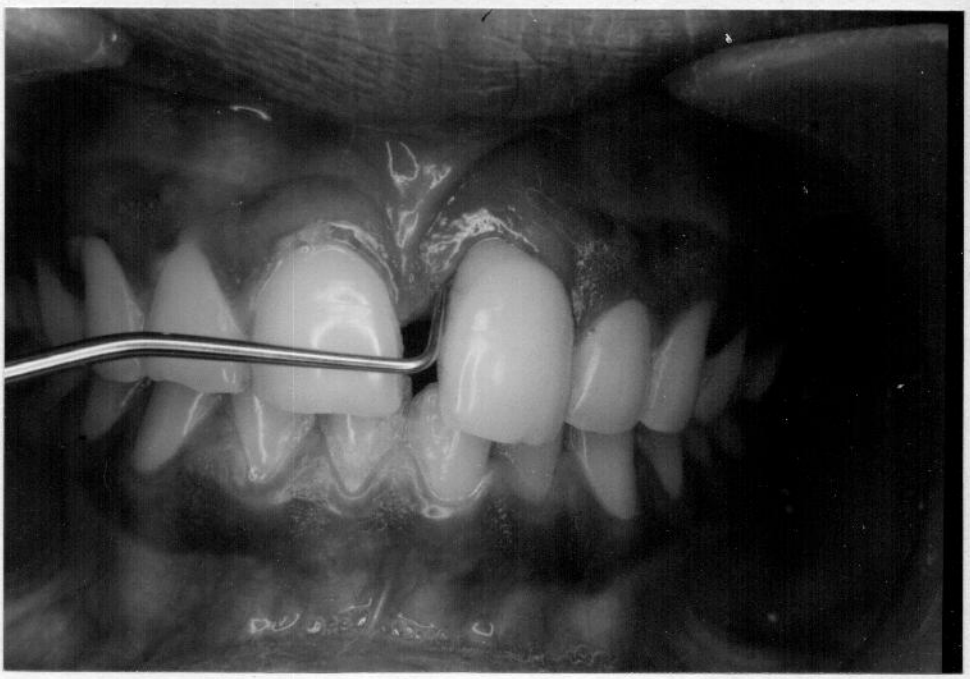


APPENDIX - 4

(a) THE ARMAMENTARIUM



(b) CPITN - 3 (Shallow Pockets)



(c) CPITN - 4 (Deep Pockets)

(d) ADULT PERIODONTITIS