Treatment Outcome of the Graduate Periodontics Clinic at the University of British Columbia. Short Term and Long Term Results.

By

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Abstract

The active treatment and maintenance for 100 periodontal patients (2,512 teeth) in the graduate periodontics clinic at the University of British Columbia was surveyed. All patients were in maintenance for at least 2 years and had an average duration of maintenance of 5.2 years. A subgroup of patients that were maintained for an average of 11 years was also investigated (19 patients). Patients and teeth were evaluated separately in order to determine the effectiveness of our treatment and maintenance program in terms of probing depth (PD) reduction, attachment level (AL) changes and tooth loss. Pockets were further divided into moderate (4-6 mm) and deep (≥7 mm), while teeth were grouped into molars and nonmolars. Additionally, prognosis of individual teeth and their long-term survivability were investigated.

It was found that PD improved significantly during active therapy for all pockets, i.e. 2.1 mm in deep pockets and 0.7 mm in moderate pockets. This improvement continued for the duration of maintenance. Attachment levels improved during initial treatment but slightly deteriorated during maintenance. However, clinically all attachment levels were stable for the duration of our study. Smokers showed similar initial attachment gain and probing depth improvement with nonsmokers. During maintenance though, both AL and PD measurements were better for nonsmokers. Smoking didn’t result in significantly greater tooth loss compared to nonsmoking patients in our study. Furthermore, if a patient was assigned in the severe periodontitis group it was likely that he/she would show a
better response in PD reduction (1.38 mm vs. 0.34 mm; p<0.01) and attachment gain (0.64 mm vs. -0.48 mm; p<0.01) compared to moderate periodontitis patients.

Pretreatment prognosis and its association with tooth survivability was also studied and the results indicate that although at 5 years the prognoses of “good” and “fair” seem to overlap, by the 11-year mark they are well separated in terms of percentage of survivability. At 5 years the percent of surviving “good” teeth is 99.4% and that of “fair” teeth is 95.6%. However, at 11 years these numbers change to 99.1% and 89.3% respectively. Notably, the “poor” teeth still have a 79% chance of surviving for 5 years. This number was similar even when evaluating the “poor” molars, alone which showed a 77% survivability. With molars though we also showed a direct association between survivability and the clinical parameters of increased mobility and furcation involvement.

These results seem to indicate that the effectiveness of the UBC graduate periodontics clinic in maintaining its patients is comparable with other studies. Tooth loss appears to be well controlled even for smokers. However, it is likely that a more aggressive smoking cessation protocol would be advantageous to our patients especially during maintenance. When assigning a prognosis we found our criteria to be reasonable and well founded. Our suggestion of using only 4 categories of prognoses instead of the usual 5, was proven to be valid and well supported at 11 years but not at 5 years. It seems that perhaps this modified prognosis assignment (with 4 categories) that we implemented may be more reasonable and effective in terms of communication with patients and with other colleagues.
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Glossary of Terms

**Accuracy:** A measurement is accurate if its performance on average is close to the true value to be determined. So, a measurement is said to be accurate if the result, expressed as a range of possible values, includes the “true” value.

**Attachment Loss:** Apical migration of the connective tissue attachment to the root surface beyond the cemento-enamel junction.

**Clinical Attachment Level:** The distance from the cemento-enamel junction to the tip of a periodontal probe during periodontal diagnostic probing.

**Chronic Periodontitis (CP):** An infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment and bone loss and is characterized by pocket formation and/or recession of the gingiva.

**Long Term (LT):** Refers to the duration of studies that are at least 5 years. In the context of this study it also refers to the subpopulation of our study sample that were maintained in our clinic for a minimum of 5 years.

**Periodontal Pocket:** An abnormal apical extension of the gingival crevice caused by migration of the junctional epithelium along the root. It is a clinical approximation of the loss of connective tissue attachment from the root surface.

**Polydipsia:** Chronic excessive intake of water.

**Polyphagia:** Excessive ingestion of food.

**Polyuria:** Excessive secretion of urine.
**Precision:** The precision of a measurement refers to how close repeated measurements of the same quantity are to each other. A measurement is said to be precise if there is only a small spread of numbers around the average value.

**Probing depth (PD):** Distance from the gingival margin to the base of the probeable crevice. It is the clinical approximation of the depth of a periodontal pocket or sulcus.

**Pruritis:** Itching.

**Resolution:** How fine a detail can be measured is known as the resolution of a measurement.

**Reliability:** The extent to which a measurement is consistent and free from error.

**Scaling & Root Planing (S/RP):** This is also collectively known as debridement and it refers to the instrumentation of the crown and root surfaces. It aims for the removal of plaque, calculus, stains and also of cementum that is rough, impregnated with calculus or contaminated with toxins or microorganisms.

**Short Term (ST):** In this text short term refers to the duration of studies that are under 3 years. Additionally, short term also refers to a subpopulation of our study sample that are maintained 2-5 years.

**Supportive Periodontal Therapy (SPT):** Procedures performed at selected intervals to assist the periodontal patient in maintaining oral health. They involve professional debridement/ removal of local irritants and they follow active therapy in sequence.

**Validity:** The extent to which an instrument measures what it is intended to measure.
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Chapter I- Introduction

The end goal of all periodontal treatment is to improve periodontal health and to provide a result that is maintainable by the patient in the long term. There have been a number of clinical trials and studies globally, investigating periodontal disease progression and tooth loss during maintenance treatment. However, there is very little information on the process of assigning a prognosis and long term tooth survivability. To date no study has been done examining tooth survivability based on their pretreatment prognosis. This is quite different than the prognosis assigned by McGuire and Nunn (1991, 1996), which was assigned after completion of active therapy and prior to maintenance.

By conducting this long term retrospective study we wanted to investigate the effectiveness of our graduate periodontics clinic in terms of controlling disease progression, and the effectiveness of our supportive periodontal program in terms of SPT frequency and disease stabilization. We also wanted to determine the long term survivability of teeth when given a specific prognosis. Additionally, we wanted to evaluate the outcome of reducing the number of prognosis categories from 5 that most studies use based on McGuire and Nunn (1991, 1996) to 4 that we use in our clinic at the University of British Columbia. This could set a new precedence in the criteria of assigning a prognosis. All our tests utilized two sets of data; averages from every patient as a whole and values from individual sites within each patient.
II.1 Periodontal Diseases

Introduction

Periodontal diseases are mostly infections of bacterial origin. By evaluating the mean annual attachment loss from longitudinal studies it seems that periodontal diseases progress slowly and continuously in all areas. However, a series of different studies in the 1980s challenged that notion and provided evidence to support a different theory, i.e. diseases of the periodontium may progress in a rather spontaneous manner at different sites at different rates and therefore leading to the necessity of using “sites” as “experimental units” rather than “patients” (Goodson et al., 1982; Haffajee et al., 1983; Lindhe et al., 1983; Haffajee et al., 1983; Lindhe & Nyman, 1984; Socransky et al., 1984; Haffajee et al., 1985; Lindhe et al., 1986; Lindhe et al., 1989).

Mode of Disease Progression

Data from clinical trials have commonly been analyzed by using the means of clinical measurements such as probing depth and attachment loss. Evaluation of the results from these studies have shown that although the mean was sensitive to small changes in large numbers of sites it was insensitive to major changes in a smaller number of sites (Haffajee et al., 1983). So when there is a overall mean “gain” of attachment in a treated
patient of e.g. 0.1 mm, it is not clear whether this mean gain has occurred uniformly at each site throughout the patient’s entire dentition, or whether certain sites exhibited significantly large gains and losses of attachment (Haffajee et al., 1985).

Goodson et al (1982) made an effort to shed some light in this research question. He measured the attachment levels of 2 sites on each tooth in 22 untreated subjects. He took measurements every month for 1 year. He found significant differences between patients and between different sites within each patient. Although the disease was untreated and if it followed the “continuous model” the affected sites should continue to lose attachment, another trend appeared. Most sites didn’t change but 5.7% of sites became significantly deeper (i.e. >2 mm of change, p<0.01) while 11.5% of sites became significantly shallower (p<0.01). In a Swedish population of untreated periodontal patients that was followed up for 6 years, only 11.6% of sites showed attachment loss over 2 mm while 50% of sites showed no measurable loss of attachment (Lindhe et al., 1983). Lindhe tested the hypothesis that sites with advanced loss of periodontal attachment would be more prone to additional destruction when left untreated but this hypothesis was not proven. These studies question the validity of using mean patient measurements to investigate the patterns of periodontal disease changes during maintenance. Lindhe and Nyman (1984) supported this with their study of a population who had “extremely advanced periodontal disease” and were maintained (3-6 month recalls) for 14 years. For example, in their study they found that deep pockets (>6 mm) showed a gain in attachment of 1.3 mm. When analyzing the data from individual patients instead of the
entire group, they discovered that there were certain sites in some patients that showed a marked loss of attachment of 5 mm or more.

This evidence directed different researchers to the theory of a different mode of periodontal disease progression other than a slowly progressive “continuous” model. Socransky et al (1984) suggested a “random burst model” (activity occurs at random at any site) and an “asynchronous multiple burst model” of destructive periodontal disease where the majority of destructive disease activity takes place within a few years of an individual’s life specifically. The concepts of “random bursts” is limited to random in regard to time and previous loss of attachment. However, predisposing factors still have to be present in those sites. Additional studies have been conducted and their results support these “burst models” (Socransky et al., 1984; Haffajee et al., 1985; Haffajee et al., 1986).

Other studies emerged that questioned the “burst” hypothesis. Some argue that the intervals may still be too long in those studies to come to a conclusion on the specific pattern of periodontal disease (Sterne et al., 1992). He actually argued that the gradual loss progression model is difficult to statistically support but on the other hand there are too many possible variables that may question the validity of the “burst” model as well. Ralls and Cohen (1986) seem to suggest something similar by criticizing the statistical analysis used to support the “burst” model. It seems that both the continuous and burst hypotheses of disease progression have merit. In summary, only a few individuals experience advanced tissue destruction around several teeth. The progression of this
disease may well prove to be a combination of a continuous model with brief episodes of localized exacerbation and occasional remission.

Conclusion

The significance of these models is that in order to appropriately monitor patients in maintenance and their disease stability (or lack of stability) it is imperative to collect and analyze data from multiple sites on each tooth within the dentition (Beck & Löe, 1993). Otherwise, by using overall mean values for groups or even an individual, we will often fail to adequately describe the progression that may occur in certain patients or certain sites. This will impact our efforts to determine microbial pathogens or host contributing and host controlling factors.
II.2 Measurement of disease

Introduction

Periodontal probing is the “gold standard” of the diagnostic instruments available to determine the presence and severity of periodontitis. It is used to assess the depth of periodontal pockets and healthy sulci, gingival inflammation and quantity of dental plaque (Silness & Loe, 1964; Loe & Silness, 1963). The World Workshop in Clinical Periodontics (1989), defined the use of calibrated periodontal probes to measure:

1) Probing Depth: Distance from the gingival margin to the base of the probeable crevice. It is a clinical approximation of the depth of a periodontal pocket or sulcus.

2) Clinical Attachment Level: Distance from the cementoenamel junction (CEJ) to the base of the probeable pocket/sulcus. It is a clinical approximation of the loss of connective tissue attachment from the root surface. Probing depth and clinical attachment levels are used as a reference (baseline) for future changes in attachment levels.

3) Relative Attachment Level: Distance from a fixed landmark (other than the CEJ) to the base of the probeable crevice. When the CEJ is not detectable or is missing due to a dental restoration, the clinical attachment level cannot be measured. In such situations, another fixed landmark such as a stent or the margin of a restoration is used (Nevins et al., 1989).
There are two ways by which error may be introduced during probing. These include the error associated with the probing technique and the instrument (probe) itself. Errors associated with the probing technique include probing error introduced by varying insertion force (Garnick et al., 1989; Van der Velden, 1978), degree of inflammatory status of the tissues (Armitage et al., 1977; Caton et al., 1981; Fowler et al., 1982; Garnick et al., 1989; Robinson et al., 1979), lack of standardized probe placement and angulation during insertion into pockets (Mintzer et al., 1993; Watts, 1987; Zappa et al., 1993), site of entry (Glavind et al., 1967; Freed et al., 1983; Khocht et al., 1998), pain felt by patient on probing (Coppes, 1972; Schmid, 1967). Errors introduced due to probe itself arise from various diameters of the probe tip (Atassi et al., 1992; Keagle et al., 1989), lack of precision of probe markings and precision with which the probe calibrations can be read (Winter, 1979; Van der Zee et al., 1991).

II.2.i Measuring attachment loss & pocket depth with periodontal probes

Acquiring the Probe Reading

DEGREE OF INFLAMMATION OF PERIODONTAL TISSUES

The average depth of the periodontal sulcus is 0.69 mm while the length of the junctional epithelium and the connective tissue attachment is 0.97 and 1.07 mm respectively (Gargiulo, 1961). The possibility that periodontal probing does not record the depth of
the anatomic sulcus or pocket was suggested by Schroeder & Listgarten (1971) who pointed out the likelihood that probing may result in tissue penetration by the probe with a consequent overestimation of actual sulcus or pocket depth. Listgarten also indicated that the extent of tissue penetration might be related to a variety of factors such as thickness of the probe, pressure applied, contour of the tooth surface (Beardmore, 1963), degree of inflammatory cell infiltration and accompanying loss of collagen fibers.

Based on a study of 116 anterior teeth and premolars with varying severities of untreated periodontal disease (1-9 mm of attachment loss), it was concluded that the probe tip reaches the most coronal level of the connective tissue attachment (Sivertson & Burgett, 1976). The clinical sulcus depth could not be related to the histological sulcus depth because the probe easily penetrated the junctional epithelium. Therefore, instead of measuring the actual pocket depth (what the probe is supposed to measure) it also included the penetration into the connective tissue fibers as well, thus reducing the validity of the measurement. The correct definition of this measurement is “probeable pocket depth” rather than simply “pocket depth”.

The relationship of the probe tip to the most apical extension of the junctional epithelium depends on the gingival tissue inflammation and to a lesser extent on the tooth type. In beagle dogs the periodontal probe stops 0.39mm short of the apical termination of the JE in the clinically healthy group, 0.10 mm short in the experimental gingivitis group and 0.24 mm past the apical termination of the JE in the periodontitis group (Armitage et al., 1977). There was also a strong correlation (r = 0.78) between probing beyond the JE and
the level of inflammatory infiltrate. Spray found an average of 0.27 mm penetration of
the probe into the connective tissue attachment in humans and also suggested that the
state of health of periodontal fibers may influence pocket measurements (Spray et al.,
1978). When comparing probe-tip penetration in treated and untreated periodontally
involved teeth (buccal aspect of single rooted teeth with PD ≥6 mm), Fowler (1982)
found an average penetration into the connective tissue attachment (beyond the JE) of
0.45 mm. With treatment, the average penetration was 0.73 mm short of the apical
termination of the JE. The difference of probe penetration between untreated and treated
specimens was approximately 1.2 mm confirming probe penetration into the connective
tissue of inflamed gingiva. In a study of 110 periodontally involved teeth it was found
histologically that the periodontal probe stopped at the first intact periodontal fibers,
penetrating the JE by an average of 0.43 mm. The probe did not penetrate the JE in only 2
out of 110 teeth (Saglie et al., 1975).

On the other hand Caton et al (1981) found no significant difference in the distance
between the termination of the probe tip and the apical end of the JE in specimens either
positive or negative for visual inflammation or bleeding after probing. However, no
information on disease severity was provided and the gingival biopsies were taken on the
midbuccal surface of the teeth whereas surgery was performed to correct interproximal
pocketing, which suggested the investigators selected the less periodontally involved sites

Smoking status of the patient can also affect probing depth measurements. The
periodontal probing measurements in the molars of heavy smokers and non-smokers were
compared and showed that the discrepancy between pre-extraction measurements and post-extraction readings (histological evaluation of extracted teeth) was smaller in heavy smokers (0.59 mm vs. 0.82 mm in non-smokers) and increased in deeper pockets (CT penetration was 1.04 mm in smokers vs. 1.78 mm in non-smokers) (Biddle et al., 2001). The authors hypothesized that this finding is due to the reduced inflammation (increased fibrotic tissue in smokers/ decreased inflammatory response). Therefore, the validity of probing measurements at molar sites in smokers is probably higher with a closer approximation to the true pocket depth and attachment levels in heavy smokers compared to nonsmokers.

From the previously mentioned studies it becomes apparent that the degree of tissue inflammation influences the precision (when comparing pre- and post-treatment readings of the same site) and the validity (mostly in diseased state) of the probing depth measurement. Probing depth measurements also show lack of reproducibility. When 3 mm, 4-6 mm, and 7 mm probing depths were multiply recorded there were significant differences of 0.5 mm, 0.7 mm, and 1.3 mm, respectively (Espeland et al., 1991). These data suggest reproducibility of probing depth measurements becomes more difficult with increased probing depths.

PROBING FORCE
Penetration of the probe increases with increasing probing force (Hassell et al., 1973; Van der Velden et al., 1978; Van der Velden, 1979). Therefore, use of a standardized force is required to obtain reproducible measurements. Using a manual probe
approximately 90% of the recordings could be repeated within ± 1 mm both for intra- and inter-examiner comparisons (Badersten et al., 1984). The level of reproducibility varied notably between patients and was improved following nonsurgical periodontal therapy. In periodontally involved teeth, when probing forces were increased from 0.15 to 0.75 N the mean probing depth also increased from 2.08 to 3.71 mm (Van der Velden & Vries, 1978). In a subsequent study of probing force & relative attachment level in molars, the mean probing depth increased from 3.97 mm with a force of 0.50 N to a depth of 5 mm when 1.0 N of force was utilized (Van der Velden, 1979). This is very important and it has been shown that every clinician has his/her own characteristic probing technique with a probing force that varies from 5-135 g, i.e. 0.05-1.32 N (1 kg=9.8 N) (Freed et al., 1983). In this same study 63.3% of the sites showed a discrepancy of ≥ 2 mm in the probing depth measurements when using 0.75 N vs. 0.15 N force. Only 43.1% of the sites continued to show this discrepancy when the comparison was between 0.75 N and 0.25 N. This difference was more prominent in deeper pockets. Therefore, force dramatically affects recorded pocket depths and its validity.

To improve the reproducibility of probing controlled-force probes have been developed (Kalkwarf et al., 1986; Khocht et al., 1998). When comparing a manual with a pressure-controlled probe in a longitudinal study of 972 teeth (over 25,000 sites) the match between their measurements declined as the probing depth increased from a 97% agreement in shallow sulci (1-3 mm) to 83% in pockets over 6mm (Kalkwarf, 1986). When comparing a manual probe, sensor-probe of 0.20 N, and electronic probe it was observed that the reproducibility of the manual and sensor probe was significantly higher
(94% within 1 mm) than that of the electronic probe (70% within 1 mm). These numbers decreased to 75% (manual), 84% (sensor-probe) and 57% (electronic) respectively in pockets over 6mm (Khocht et al., 1998).

AREAS OF DENTITION PROBED

Significant differences with regard to the tooth surface and sextant of the dentition to be probed are also present. Hassell et al (1973) demonstrated that clinicians exerted 0.3 – 0.53 N (3.0-54.0 g) of force in the anterior region and up to 1.37 N (140.0 g) in the posterior region of the mouth. Errors of probing are found between anterior and posterior areas but differences are also found on specific areas of teeth. Distal surfaces are probed with the greatest force followed by the mesial surfaces while the lowest forces are applied on the facial surfaces. The forces used to probe the posterior teeth are significantly greater than those used in the anterior segments (Freed et al., 1983). When comparing interproximal measurements taken in the posterior teeth between a manual probe and a pressure-controlled probe (0.5 N of force), the manual probe produced consistently deeper readings. However, in the anterior region the manual probe produced consistently shallower readings, thus confirming that more force is generally applied in the posterior region (Kalkwarf et al., 1986). Impaired vision, intensity of retracting tissues, and compromised instrument position may reduce tactile sense in the posterior sextants of the dentition and explain the different forces required by examiners.
Errors leading to an underestimation of pocket depth could result from poor angulation of the probe in relation to the periodontal pocket. Malposition of the probe may be due to anatomic features such as crown contour or interdental relationships (Listgarten, 1972). However, there is a leeway in the angulation that can be accurately used. Angulating a probe 25° from the long axis of a non-restored tooth resulted in a discrepancy of only 0.5mm from measurements taken in the direction of the long axis of the tooth (Ziegler & Allen, 1978). There is also a horizontal component to probe placement. When examining the effect of variation of horizontal positioning of the probe on pocket depth readings, it was found that a substantial proportion of altered repeat probing measurements might be explained by variation in the transverse plane (Watts, 1989).

The Probe Itself

Although manufacturing methods used to produce periodontal probes have been improved in recent years, probe-to-probe variations in calibration markings up to 0.5mm still occur (Van der Zee E et al., 1991).

DIAMETER OF PROBE TINE & TIP

Van der Zee et al (1991) investigated 7 different probe types regarding their tine and tip diameter. With one-way-analysis of variance he revealed highly significant differences in tine diameter (in some of the tapered probes) as the diameter gradually increased away from the tip. The probe tip diameter also ranged significantly from 0.28 mm (Michigan
"O") to 0.7 mm (Williams). Therefore, tine diameter influences probing measurements.

From a study aimed to identify optimal tip diameter, Keagle JG (1989) concluded that 0.6 mm was the most discriminatory of health/disease status.

The effect of tine diameter on probing pocket depth measurement between parallel and tapered probes was examined by Atassi et al (1992). All tines were mounted on a calibrated pressure-sensitive handle using 0.25 N force. The parallel-sided tines had an actual mean diameter of 0.46 mm and increased only to 0.52 mm at the 10 mm marking. The mean diameter of the tapered tine tips was 0.48 mm and increased to a mean of 0.72 mm at the 5 mm marking, and to 0.98 mm at the 10 mm marking. Therefore, probe tine & tip diameter influence the validity of probing depth measurements, with the parallel tine having a tendency to yield greater probing depth with deeper pockets.

ACCURACY OF PROBE MARKINGS

In a classic study, Winter (1979) examined 129 manual periodontal probes that he collected from different private practices. Out of 387 total measurements, only 130 were accurate. Time of manufacturing was correlated to the probe’s accuracy. When testing the accuracy of the 7.0 mm mark he found that half of the “old” Williams probes were actually between 7.4-7.6 mm while the range extended from 6.8 to 7.9 mm. The “newer” Williams & Michigan probes showed a high degree of precision, but were not always accurate. Twelve years later in a different study, Van der Zee et al (1991) investigated the width of markings and accuracy of calibration from probe tip in 7 different probe types using stereomicroscopy (magnification x40). The authors found that the markings
themselves varied from having no measurable width for engraved bands to a maximum width of 1.13 mm for painted bands. Painted bands were found to have a mean width of 0.70 mm. Irrespective of probe type engraved bands were found to be the most accurate. As for the marking position on the probe tine, in very few cases was a marking exactly coincident with the manufacturer's designated calibration to within 0.01 mm. It becomes obvious that even 12 years after Winter's study, although the accuracy and precision of most probe types have improved significantly, there is still a small error in probe markings. However, this remaining error likely has no significant clinical implications but it may affect the accuracy of measurements.

II.2.ii Measuring furcations with probes

Molars are lost more often than any other tooth type (McFall, 1982; Wilson, 1987). One significant reason that may explain this loss is the presence of furcations. The extent of the periodontal destruction between the roots influences the prognosis of the involved teeth. In addition, the severity of furcation involvement is often used as a basis for treatment planning in periodontal therapy (e.g. surgery vs. extraction) (Zappa et al., 1993). Hamp et al (1975) introduced an index for classifying the depth of horizontal involvement in 3mm increments to describe depth of probe penetration in 3 degrees. The reference point is an imaginary plane drawn across the convexities of the root surfaces forming the furcation. This index is currently used in most University dental clinics including UBC's graduate and undergraduate dental clinic and includes 3 classes; Class I
(horizontal attachment loss up to 3 mm), Class II (over 3 mm but not through-and-through) and Class III with a remaining through-and-through defect.

When actually comparing the reproducibility and validity of 3 different probes Eickholz (1998) found the Nabers probe (color-coated probe with 3 mm increments) to provide excellent agreement when assessing buccal, lingual, and mesiolingual furcations, but only moderate for distolingual furcations. However, the Nabers probe was consistently better than the straight flexible and straight rigid probe. Generally actual furcation depths are underestimated. When there was a true through-and-through furcation involvement (as shown after surgical exposure), 27% of the times the clinician had categorized it as either class II or class I using the Hamp Index and nearly 10% of originally class II furcations were proven to be class III (Eickholz et al., 1998; Zappa et al., 1993).

**Conclusion**

The periodontal probe still remains the most important clinic tool used by dentists to collect data needed to make a diagnosis and prognosis of the dentition. Errors may be introduced by the clinician, the manufacturing process and the existing condition of the periodontal tissues. The probe tip may fall short of the junctional epithelium in a healthy site and may pass well beyond the junctional epithelial attachment in an inflamed pocket. Presence of gingival inflammation results in probe penetration beyond the histological pocket by 0.45 mm in shallow pockets up to 1.78 mm in the deeper pockets. Resolution of the inflammation alone after treatment results in approximately 1.2 mm of probable
pocket depth reduction when compared to pre-treatment measurements. The force with which the clinical probes also affects the position of the probe tip. There is a significant range of pressures applied by clinicians (0.05 N to 1.35 N) although the ideal suggested force is 0.25 N. When moving from the anterior to the posterior dentition the same examiner increases his/her probing force by 4 times. The probe itself may be parallel vs. tapered with the former favoring deeper probing values. The probe markings may result in small errors as well depending if they are painted bands or engraved. Intra- and inter-examiner reproducibility usually is within ± 1 mm in 90% of the cases. Regardless of possible errors that may be associated with probing it is still a critical instrument that is required for patient assessment. Errors are inherent in its use and collectively this affects the accuracy of the data that is collected.
II.3 Risk factors that may influence the initiation or progression of periodontal disease

Introduction

Risk factors that are linked to periodontal disease include smoking, diabetes and genetic influences. It is known that 22% of the Canadian population over the age of 15 are current daily smokers with an average of 16 cigarettes/day (Statistics Canada, 2003). Cigarette smoking is associated with many systemic complications/diseases and is also known for its detrimental effects on the oral tissues. Halitosis, stained teeth, tooth loss, bone loss, oral cancer and decreased success of dental implants are some of them. With over 4,000 toxins and carcinogens in smoke, there are several effects on the periodontal tissues and periodontal disease initiation and progression. Diabetes mellitus (DM) is a group of disorders resulting from the malfunction of insulin dependent glucose homeostasis and also involves impaired lipid and carbohydrate metabolism. Type 2 diabetes is most prevalent; approximately 90% to 95% of people diagnosed with diabetes in the U.S. have Type 2 diabetes. (Taylor et al., 2001). Periodontitis is a multifactorial inflammatory disease that is initiated and perpetuated in the presence of specific bacteria. Although bacteria are essential for initiation of periodontitis, the virulence & pathogenicity of bacteria alone is not sufficient to explain all the differences in diseases severity that are seen clinically. There is evidence that in many common chronic diseases there are host modifying factors that may amplify specific mechanisms of the disease and therefore lead to a more severe clinical manifestation (Kornman KS & Di Giovine FS,
Cytokines have been shown to play a crucial role in the pathogenesis of many infectious diseases including periodontal disease (Genco RJ, 1992).

II.3.i Smoking

**Historical Studies**

Solomon et al (1968) evaluated smokers and nonsmokers and found gingival recession and alveolar bone loss to be higher in smokers. Periodontal disease as measured by the PDI (Ramfjord’s Periodontal Index) was more severe in smokers (Summers & Oberman, 1968). However, in neither of these two studies was oral hygiene considered. This likely had a significant impact on the result because staining and calculus were found to be present in significantly higher quantities in smokers compared to nonsmokers (McKendrick et al., 1970; Sheiham, 1971; Preber et al., 1980; Preber et al., 1985). When smokers and nonsmokers with the same oral cleanliness were compared the difference between them was not significant. This challenged the concept that smoking may be an actual risk factor and suggested differences in disease severity between smokers and nonsmokers could be attributed to the lower oral hygiene effectiveness in smokers.

**Contemporary Views**

In the evaluation of a representative sample of the United States population as part of the National Health and Nutrition Examination Surveys, Ismail et al (1983) using a multiple
linear regression model, evaluated the relationship of smoking to periodontal disease in 3000 individuals. All mean values obtained were adjusted for potential confounding variables such as age, sex, race, socioeconomic status, oral hygiene and frequency of tooth brushing. In this study, smokers had significantly higher mean PI scores in all age groups (25 to 74 years) and significantly higher scores in the debris, calculus and oral hygiene indices. Smoking was shown to have an independent direct association with periodontal disease although less strong than the association of oral hygiene with periodontal disease. Smoking may also have a negative effect on periodontal tissues even in the absence of periodontal disease. Smoking was associated with attachment loss and/or recession in individuals with minimal or no periodontal disease (Gunsolley et al., 1998). Smokers had marginally (but statistically significant, i.e. P<0.05) more plaque and gingival inflammation than nonsmokers but smokers had significantly greater recession and/or attachment loss than nonsmokers. Confounding variables like age, sex, plaque, and calculus, have been accounted for and the odds of having a mean probing depth ≥ 3.5 mm were 5 times greater (odds ratio: 5.3) for smokers than the nonsmoker subsample (Stoltenberg et al., 1993).

Clinical Impact of Smoking on Periodontal Disease Initiation and Progression

BONE LEVELS & POCKET DEPTHS

Bergstrom studied the differences between bone levels of smokers and nonsmokers with similar levels of good OH. The smokers group smoked on average 14 cig/day for 22 years. There was consistently lower remaining bone height percentage associated with the
smoker group and the combination of smoking with plaque had a cumulative effect on bone loss (Bergstrom & Eliasson, 1987). In addition, the distance between the CEJ and the interdental septum was examined in smokers and nonsmokers (both with good oral hygiene), and bone loss was positively correlated with smoking (Bergstrom et al., 1991).

ATTACHMENT LOSS AND TOOTH LOSS

The association found between smokers and deeper probing depths was confirmed and over 80% of the sites with at least 6mm of attachment loss were found in individuals who smoked (Linden and Mullally, 1994). In addition the long-term tooth survivability seems to be affected by smoking. Holm (1994) investigated 273 subjects twice in 10 years for tooth loss and found that 93 subjects (34%) lost 130 teeth over the course of 10 years due to periodontal complications. In each age group tooth loss was significantly higher in smokers. Heavy smokers (> 15 cig/day) in the younger groups (30-50 years old) had a relative risk for tooth loss that was 4.55 greater when compared to nonsmokers from the same group. The combination of poor OH and heavy smoking showed additional detrimental effects on tooth survivability.

In a cross-sectional study of 705 adults current cigarette smokers had the highest prevalence of moderate and severe periodontitis (25.7%) compared to non-smokers (13.1%). There was a positive correlation with the number of missing teeth with current smokers and nonsmokers having 5.1 and 2.8 missing teeth respectively; the more the patient smokes, the greater the degree of periodontal disease that will be found (Albandar et al., 2000).
RESPONSE TO TREATMENT

Smoking appears to affect periodontal treatment effectiveness. There was no difference between smokers and nonsmokers when gain in attachment was evaluated after initial non-surgical treatment (mean clinical attachment gain was 0.52 mm vs. 0.50 mm in favor of nonsmokers), but there was a significant difference in the mean probing depth reduction in favor of the nonsmokers (1.14 mm vs. 0.76 mm). Therefore, even in the presence of antimicrobials (that the authors used), smoking still has a significant unfavorable effect on probing depth reduction (Kinane & Radvar, 1997). When examining deep pockets it was found that the difference in pocket depth resolution between smokers and nonsmokers was greater in favor of nonsmokers after root planing and this difference increased as the pocket depth increased (Grossi et al., 1996). In addition a significantly greater percentage of pockets exhibit probing depth reduction in the nonsmokers group when compared to smokers: 71% vs. 59% at 4 mm and 91% vs. 73% at 6 mm. Even after a modified Widman flap procedure (without osseous resection) heavy smokers show a reduction of 0.76 mm vs. 1.27 mm (in nonsmokers) in probing depth in the presence of moderate-severe periodontitis (Preber & Bergstrom, 1990).

In a study in Minnesota the authors evaluated the effects of PMN defects on refractory patients. While the quality of oral hygiene and access to dental care were high, a retrospective search for associated environmental variables showed that 90% (28 of 31) of the refractory patients were smokers. The frequency of smokers is particularly striking, since only 21% of adults in Minnesota use tobacco regularly. This study suggests a
positive association between a history of smoking and refractory periodontitis (MacFarlane et al, 1992).

ETHNICITY

Race may also play a role on the measure of detrimental effect smoking has on periodontal tissues. Qiunn et al (1998) established degree of smoking based on serum cotinine levels (a stable metabolite of nicotine). Nonsmokers had low cotinine levels <20 ng/ml compared to ≥75 ng/ml for smokers. They found white smokers had significantly more teeth with >2 mm of attachment loss compared to black smokers (78% of teeth vs. 68%) and significantly more teeth having >5mm of attachment loss (30% of teeth vs. 21%). When comparing between smokers and nonsmokers within each race they found that smoking had a significant effect on attachment loss only in white patients with chronic periodontitis but no significant effect on clinically similar black patients. Further studies have to be performed in order to determine if white subjects who smoke suffer more detrimental effects than blacks.

Smoking Impact on Microbiological Status of Patients

The effect of smoking on the microbiological profile associated with periodontal disease is less clear. For example when the prevalence of periodontal disease associated pathogens, Porphyromonas gingivalis, Actinomyces actinomycetemcomitans (A.a.), Prevotella intermedia (P.i.), Eikenella corrodens (E.c.), Fusobacterium nucleatum (F.n.), Bacteroides forsythus (B.f.), Treponema denticola (T.d.), were examined by cell culturing
or polymerase chain reaction (PCR) there was no difference between smokers and nonsmokers (Stoltenberg et al., 1993; Darby, 2000). In contrast it has also been shown in a study of 1,426 subjects that current smokers were 3.1 times more likely to be infected with A.a. and 2.3 times more likely to be infected with B.f. than former smokers or nonsmokers (Zambon et al., 1996). Due to this study’s cross-sectional design no causal relationship could be determined. The ability of root planning to reduce the number of periodontal disease associated pathogens may be impacted by smoking history. Eighty eight percent (88%) of nonsmokers no longer harbored Porphyromonas gingivalis (P.g.) compared to only 33% of smokers after root planing. The proportions of smokers who became negative for B.f. was also significantly less than nonsmokers, i.e. 21% vs. 50% (Grossi et al., 1996). Smoking is positively related with the detection of cluster bacteria (i.e. B. forsythus, P. micros, F. nucleatum, C. rectus), with the odds ratios increasing with an increase of the total percent of cluster bacteria (Van Winkelhoff et al., 2001). Detection of ≥20% P.m./F.n. in treated patients was strongly associated with smoking (odds ratio: 13.8).

When comparing smokers and nonsmokers with equal levels of oral cleanliness, P.g. and P.i. were more likely to be isolated with increased probing depth in nonsmokers. Smoking increased the likelihood of culturing P.g. and P.i. in shallow sites (Eggert et al., 2001). Therefore, in the shallow sites it seems that the composition of the microflora can be different between smokers and nonsmokers. When examining 4 ethnic groups for the presence of putative periodontopathogens (molecular analysis), current smokers displayed an increased risk for harboring Treponema denticola in periodontal pockets.
with an odds ratio of 4.61. On the other hand there was a reduced odds ratio of 0.34 of T.d. in the African-American group. Race/ethnicity appeared to be a significant explanatory variable (Umeda et al., 1998).

Effects of Smoking on Gingival Arterial Blood Flow

Gingiva is an end-arterial organ with metabolic needs supplied by small-diameter blood vessels. Gingiva is at the interface of the internal (tissue) and external (sulcus/pocket) environment so it is quite possible that the host response to local microorganisms associated with subgingival plaque biofilm may be modified by the adequacy of the local blood supply.

One of the first studies to show this was conducted on rabbits by Clarke & Shephard (1984) who investigated the effects of stress (epinephrine) and nicotine on the gingival tissues. Their findings suggested that epinephrine and nicotine could synergistically constrict the end-arterial vessels of rabbit gingiva. They hypothesized this decrease in the gingival blood flow may be below the optimal level required for full and adequate protection of the periodontal tissues and this may affect the balance of the host-parasite relationship in favor of disease. Although Clarke and Carey (1985) advocated that blood flow is severely reduced in the gingival crest in humans for 2-3 hours after 1 cigarette, they failed to present supporting data. In contrast, it was found that relative blood flow to the gingiva (GBF) increased quickly by 25.4% above resting values and remained relatively high during the first 5 minutes after smoking (17.6%). It returned toward
baseline within 10 min but never fell below baseline levels afterwards (Baab & Oberg, 1987).

**Immunological Effects**

IgG2 is the major immunoglobulin subclass that reacts with bacterial carbohydrates and lipopolysaccharides and serves as an opsonin. Consequently, an impairment of IgG2 responses may increase the risk of bacterial infection including periodontal diseases. When Caucasian subjects smoked (regardless of the presence of periodontitis or not) their serum IgG2 levels decreased significantly from 3860 µg/ml to 2804 µg/ml vs. 4197 µg/ml to 2702 µg/ml for the healthy subjects (Quinn et al., 1998). The dramatic reduction in serum IgG2 seen in smokers is associated with an increased severity of disease status (Tangada et al., 1997).

II.3.ii Diabetes mellitus

**Epidemiology**

An estimated 12-14 million individuals in the United States (U.S.) have diabetes with about half of these people being aware of their disease. Over 200 million people globally are believed to have DM. Close to 90% of these cases have Type 2 DM with the rest being Type 1 or having diabetes associated with pregnancy or a systemic disease (Position paper, J Perio, 1999). Additionally, approximately 600,000 new cases are
diagnosed each year (Yalda et al., 1994). Epidemiological studies also show the impact of the DM associated complications in the general population. DM is the third leading cause of death in the U.S. and accounts for about 40,000 deaths annually. The relative risk of diabetics acquiring end-stage renal disease is 25 times that of nondiabetic individuals. A diabetic is also 20 times more likely to become blind. Ultimately, DM has a severe impact on the life expectancy of the diagnosed individual. This negative effect is dependant on how early the diagnosis was made. If someone is diagnosed with DM at the age of 10 he is expected to live 17.2 years less than the average life expectancy while if the diagnosis is made at the age of 30 he will live an estimated reduced life time of 12.4 years (Shlossman et al., 1990).

Pathophysiology and Diagnosis

Glucose is the most important stimulus for insulin secretion. Insulin remains in circulation for only several minutes ($T_{1/2}= 4-8$ min) and then interacts with target tissues and binds with cell surface insulin receptors, which in turn activate glucose transport proteins. Impaired function of this insulin dependent system results in glucose accumulation in the tissues and circulation (hyperglycemia).

Insulin is stored and released from the pancreatic $\beta$-cells and has 4 major actions:

1- Glucose transfer from blood to insulin-dependent tissues, i.e. fat, muscle & liver.
2- Stimulation of transfer of amino acids from the circulation into the cells
3- Stimulation of triglyceride synthesis from fatty acids
4- Inhibition of triglyceride breakdown for mobilization of fatty acids.
Hyperglycemia may lead to microvascular complication through the increased accumulation of advanced glycation endproducts (AGEs). An increase in intracellular glucose leads to an increase in sorbitol (through the aldose reductase pathway), which in turn affects glomerular and neural tissue functions and thus may lead to complications such as neuropathy, retinopathy, and nephropathy. Another very common complication is altered wound healing. Hyperglycemia leads to increased secretion of glucose in the urine, which is now increased in volume. This increased fluid loss may lead to dehydration and loss of electrolytes. In addition, the lack of glucose utilization may lead to cell starvation (excluding CNS cells) and make the patient increase his intake of food.

The classic signs and symptoms of DM include the known triad of polyuria, polydipsia, and polyphagia, along with other likely symptoms like pruritis, weakness, fatigue, dry mouth, and repeated infections. However, these signs can only indicate the possibility of DM. The diagnosis is based on lab tests that have to confirm the physician’s suspicion on two different occasions.
### Diagnostic Criteria for Diabetes Mellitus

Diabetes mellitus may be diagnosed by any one of 3 methods. Whatever method is used must be confirmed on a subsequent day by using any 1 of the 3 methods.

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<td>1.</td>
<td>Symptoms of diabetes plus casual (non-fasting) plasma glucose $\geq 200$ mg/dl. Casual glucose may be drawn at any time of day without regard to time since the last meal. Classic symptoms of DM include polyuria, polydipsia &amp; unexplained weight loss.</td>
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| 2. | Fasting plasma glucose $\geq 126$ mg/dl. Fasting is defined as no caloric intake for at least 8 hours.  
1. FPG $< 110$ mg/dl = normal fasting glucose  
2. FPG $\geq 110$ mg/dl & $< 126$ mg/dl = Impaired fasting glucose  
FPG $\geq 126$ mg/dl = provisional diagnosis of diabetes (must be confirmed on subsequent day) |
| 3. | 2-hour post-prandial glucose $\geq 200$ mg/dl during an oral glucose tolerance test. The test should be performed using a glucose load containing the equivalent of 75 grams of anhydrous glucose dissolved in water. (This method is not recommended for routine clinical use.)  
1. 2hPG $< 140$ mg/dl = normal glucose tolerance  
2. 2hPG $\geq 140$ mg/dl & $< 200$ mg/dl = impaired glucose tolerance  
3. 2hPG $\geq 200$ mg/dl = provisional diagnosis of diabetes (must be confirmed on subsequent day) |

One of the most commonly used tests to determine the blood’s glucose levels is the glycated hemoglobin test used as a monitoring or screening tool. This test measures the amount of glucose irreversibly bound to α-hemoglobin (HbA1c) and can be used to monitor the effectiveness of diabetes control. This value is proportional to the blood glucose levels, and gives a measure of the blood glucose status over the half-life of the red blood cells, or 30-90 days. Therefore, this test gives us an indication of the blood glucose levels over the preceding 2-3 months.
There are 2 glycated hemoglobin tests available: the hemoglobin A1 (HbA1) test and the hemoglobin A1c test. The normal value for the HbA1 test is $\approx 8.0\%$ while the normal value for the HbA1c test is $\approx 6.0-6.5\%$.

**Diabetes and Periodontitis**

Several studies for the past few decades have tested the relationship between diabetes and periodontal destruction. Although there is now strong evidence of a bidirectional pathway of effect between these two chronic diseases this section will focus more on the alterations to the periodontium as a result of diabetes mellitus.

Already in the 1960s there was evidence of a different periodontal response to similar local factors between diabetics and nondiabetics (Benveniste et al., 1967; Burns, 1969; Glavind et al., 1969). A more pronounced response was especially noticed in the diabetic 30-40 year old age group. But significant differences in attachment loss were found when patients with less than and more than 10 years of disease (diabetes) were compared. Therefore, an indication of both periodontal effects of diabetes was suggested along with a positive correlation (Glavind et al., 1968). On the other hand, Hove & Stallard (1970) attributed whatever differences between diabetics and healthy people to differences in levels of local factors. In this study a 2h post-prandial blood sugar test was used with an upper diagnostic limit of 100 mg/dl, which is half of what is considered diagnostic by today’s standings (200 mg/dl). This must have resulted in a significant number of “false positive” diabetics in his study. Regardless, young diabetics (19-25y.o.) showed an
increased number of gingival inflamed units with pockets of 4-5 mm. The correlation between the metabolic control of diabetes and the frequency of inflamed gingival units was found to be weak, i.e. \( r = 0.33 \) (Rylander et al., 1987). Due to the young age of the subjects in this study (teenagers) it would be unlikely to observe a significant difference in the presence of 5mm pockets when compared to their healthy counterparts.

The Pima Indians from the Gila River Indian Community in Arizona has the world’s highest reported incidence and prevalence of Type 2 diabetes. Fifty percent (50%) of those over the age of 35 have DM. When assessing deep pockets of \( \geq 6 \) mm, it was found that 50% of the DM patients had at least 1 sextant with deep periodontal pockets (\( \geq 7 \) mm) vs. 17% of the nondiabetics (Bacic et al., 1988). When assessing rate of progression it was found to be 2.6 times higher in the DM group compared to the nondiabetics group (Nelson et al., 1990). Diabetics have been shown to exhibit an increased rate of progression of periodontal disease (38% increase of attachment loss vs. 9% in nondiabetics over 2 years) and in the presence of less local deposits (Cohen et al., 1970). The odds ratio for diabetics to have periodontal disease was 2.81 vs. healthy subjects while they also had an increased risk of 3.43 of losing interproximal bone (Emrich et al., 1991). Poorly controlled Type 2 DM patients have a significantly higher risk of alveolar bone loss than nondiabetic subjects; odds ratio is 11.4 (Taylor et al., 1998). Sznajder et al (1978) also found 34% more attachment loss in diabetics over the age of 30 when compared to nondiabetics.
Tervonen & Oliver (1993) were the first to use multiple glycosylated hemoglobin measurements (HbA1c) over a period of years to determine the long-term metabolic control of patients and its effect on periodontal disease. The important finding of this study was that all diabetics (regardless of good, moderate or poor control) had minimal periodontitis in the absence of local deposits. However, when calculus was present, poorer metabolic control was associated with increased periodontitis. Over a 5-year period of investigation in a population where the metabolic control was monitored with HbA1c, the diabetic group lost significantly more attachment 1.11 mm compared to the nondiabetic population which lost only 0.22 mm (Firatli, 1997). In regards to the loss of alveolar bone DM patients with good long-term metabolic control are not at greater risk than nondiabetics (Tervonen et al., 2000). Even the difference in the response to comprehensive periodontal treatment and long-term maintenance (5 years) between diabetics and nondiabetics was no different when the diabetic subjects were well controlled (Westfelt et al., 1996). Therefore, diabetes mellitus does not induce periodontal disease but rather serves as a predisposing factor, which can accelerate plaque induced periodontal destruction.

Pathogenic Mechanisms on Periodontal Tissues

Chronic hyperglycemia may directly or indirectly affect the periodontal tissues. Two possible mechanisms have been suggested; the polyol pathway and the production of advanced glycation endproducts (AGEs). The first one involves the enzymatic transformation of glucose to sorbitol, which may act as a tissue toxin. The second
mechanism (which has been extensively studies) is the non-enzymatic glycation and oxidation of proteins and lipids. These altered proteins include collagen, hemoglobin, plasma albumin, lens proteins, lipoproteins, etc. These AGEs accumulate in the plasma, tissues and gingiva of diabetics (Soskolne et al., 2001; Lalla et al., 2001). Glycation of collagen due to hyperglycemia results in increased thickness of the basement membrane (type IV collagen), which in turn leads to reduced metabolic waste removal within the tissues. Glycation of type I collagen (gingiva, PDL, bone) results in diabetes-induced reduction in collagen solubility (reflecting excessive collagen crosslinking), which also reduces solubility and metabolic turnover. These effects are detrimental to periodontal health (Yu et al., 1993).

In addition to changes in the glycation of the extracellular matrix, diabetics also show changes in cell function. Monocytes, macrophages and endothelial cells within the gingiva possess specific receptors for the advanced glycation endproducts, i.e. RAGEs. The properties of the cells that possess these receptor alter markedly when AGEs are bound to them. It leads to an excessive production and release of proinflammatory cytokines (mostly IL-1β, IL-6, PGE2 and TNF-α), predisposing the tissues to breakdown when stimulated by bacterial LPS. The cytokines in return may activate other inflammatory cells such as T- and B-lymphocytes. Therefore AGEs present in diabetic gingiva may be associated with a state of enhanced oxidant stress, leading to accelerated tissue injury (Schmidt, 1996).
II.3.iii  Genetic influences

Proinflammatory Cytokines

Cytokines are molecules released by host cells into the local environment and provide molecular signals to other cells thereby affecting their function. Each cytokine has one or more target cells with specific high affinity receptors. The following cytokines and inflammatory mediators are most consistently associated with periodontitis (Gemmell & Seymour, 1994; Alexander & Damoulis, 1994):

Interleukin 1 (IL-1) is a multifunctional cytokine that is one of the key mediators of the body's response to microbial invasion, inflammation, immunologic reactions, and tissue injury. It is produced by fibroblasts, epithelial cells but predominantly by macrophage-monocytes. These molecules promote bone resorption & inhibit bone formation, inhibit alkaline phosphatase activity of the PDL fibroblasts, stimulate PGE₂ release by monocytes & fibroblasts, stimulate release of MMPs that degrade proteins of the ECM, and participate in many aspects of the immune response. They participate in connective tissue destruction, leading to loss of attachment. IL-4 inhibits the secretion of IL-1, TNF-α, IL-6 and PGE₂ from macrophages and its absence is favorable to destruction of periodontal tissues. IL-6 stimulates the formation of osteoclasts and the activation of osteoclastic bone resorption. IL-8 is a chemoattractant mainly produced by monocytes in response to LPS, IL-1 or TNF-α. In periodontal tissues IL-8 is mainly found in the JE, and GCF and may attract and activate neutrophils and MMPs, which can promote tissue
destruction. TNF-α is a proinflammatory cytokine produced mainly by macrophages after stimulation with LPS and is abundant in the gingival tissues of moderate-severe periodontitis patients. It plays an important role in connective tissue destruction and bone resorption. Interferon-γ (IFN-γ) also belongs to the cytokine group and is a potent inhibitor of IL-1, TNF-α, and TNF-β stimulated bone resorption and could act as a protective factor (Van Dyke et al., 1993; Alexander et al., 1994; Academy Reports, 1999).

Role of Genetics in Periodontal Tissues

TNF-α and IL-1β can induce bone resorption and play an important role in the pathogenesis of periodontal disease. Their presence is significantly increased in severe chronic periodontitis (CP) patients compared to mild CP patients and healthy controls (Galbraith et al., 1997). Studies in twins have indicated the strong likelihood that a proportion of the clinical characteristics of periodontitis may be explained by genetic factors (Michalowicz, 1994). Some genetic variations (polymorphisms) are commonly found in humans and may represent a mechanism by which individuals exhibit variations in the manifestation of periodontal lesions.

Recent studies investigated the relationship between genetic variations (IL-1β variations) associated with cytokine production and periodontitis severity (Kornman & Di Giovine, 1998). Kornman first suggested a genetic marker associated with the severity of periodontitis in non-smoking adults. This marker (composite genotype comprising the IL-
1A\textsuperscript{889} polymorphism & the IL-1B\textsuperscript{3953} polymorphism of the gene) is associated with a 4-fold increase in IL-1 production in nonsmokers (Kornman KS, 1997). This composite genotype was present in 78% of severe patients vs. 16% of mild (Kornman et al., 1997). The frequency of the IL-1\(\beta\textsuperscript{3953}\) genotype is significantly increased in Caucasian patients with severe chronic periodontitis when compared to mild cases. This suggests that the IL-1\(\beta\textsuperscript{3953}\) may predispose an individual to increased severity of periodontal disease (Gore et al., 1998; Galbraith et al., 1999). There is a 3.6-fold higher mean concentration of IL-1\(\beta\) in the gingiva of positive genotype patients and a 2.5-fold increase in IL-1\(\beta\) production in the GCF in shallow pockets ($\leq$4mm). No significant differences were present in the deeper pockets (Engebretson et al., 1999). Additionally, the composite genotype (+) patients have an increased risk of tooth loss from periodontal disease by 2.66, which increases to 7.7 when combined with heavy smoking (McGuire & Nunn, 1999).

IL-1 genotype (+) nonsmokers are at higher risk (odds ratio of 3.75) of having moderate-severe CP compared to IL-1 genotype (-) subjects. When only the European subjects were concerned the Odds Ratio increased to 5.27 (McDevitt et al., 2000). No significant differences in the IL-1\(\beta\) prevalence between African-American localized aggressive periodontitis cases and controls were found (14% vs. 8%). This suggests that the composite IL-1 genotype associated with the severity of chronic periodontitis in Northern European nonsmokers, may not have the same association in African-American localized aggressive periodontitis patients (Walker et al, 2000). This is also applies to subjects of Chinese heritage. The prevalence of the IL-1 composite genotype is 2.3% (7 subjects) compared to that reported for Caucasians, i.e. $\approx$36% (Armitage et al, 2000).
**Role of HLA in Periodontal Disease**

HLA will only be briefly mentioned in this text. A mother, son and daughter who were diagnosed as having “periodontitis as a manifestation of systemic diseases, associated with hematological disorders” were examined. After evaluating their immunological profiles of host defence functions and human leukocyte antigen (HLA) properties it was concluded that all subjects had the same genotype, HLA-DQB1*0601 which lead to depressed neutrophil chemotaxis which in turn may have been a significant risk factor for their periodontal disease predisposition (Okada et al., 2002). Based on modern DNA techniques an association of HLA to both rapidly progressive periodontitis and chronic periodontitis has been shown. Certain HLA alleles seem to be associated with susceptibility or resistance to periodontitis in general (Machulla et al., 2002). Differences in prevalence and in the extent of attachment loss between males and females have suggested that gender-dependent HLA deviations could play a role in individual predisposition to periodontitis. Reichert et al (2002) found that gender is a confounding variable, which should be considered in further studies of HLA and periodontitis. Along with the above studies, others have been performed and all lead to the suggestion that HLA antigens should be considered as risk factors for periodontitis.

**Conclusion**

The risk factors of smoking, diabetes and IL-1β can all act alone or in combination. Although earlier studies tended to attribute whatever differences in disease severity between smokers and nonsmokers to differences in oral hygiene, recent studies have
confirmed smoking to be a true risk factor for periodontal disease. It is now known that smoking (and more so heavy smoking) negatively impacts pocket depth, progression of disease and tooth loss. AGE mediated events are critical to the pathogenesis of diabetic complications contribution to periodontal destruction. Especially when the diabetic patient is poorly controlled the elevated AGE production makes him/her increasingly susceptible to acceleration and aggravation of periodontal disease when present. It seems that the deleterious effects of the accumulation of AGEs can lead to the strong association that has been observed between diabetes (mostly poorly controlled) and the different presentations of periodontal disease.

Knowledge of factors, such as smoking, diabetes and the IL-1 genotype that are significantly associated with more severe periodontitis, should enhance the clinician's ability to estimate the future course of disease for a specific patient. It becomes more and more apparent that although the bacteria cause the disease, the individual’s genetic makeup and environmental influences, may determine how severe the disease will be. Additionally race may also come to play a role the extent of the effect risk factors may have. However, removing plaque & calculus and keeping it at low levels is sufficient to maintain periodontal health in the vast majority of individuals at risk (more so in nonsmoking subjects).
II.4 Maintenance of Periodontal Patients

Introduction

Evaluation of periodontal treatment effectiveness is based on clinical re-evaluation of pocket depth and other clinical parameters at completion of active therapy. Long-term maintenance studies determine if current treatment modalities and maintenance regimens are effective at maintaining periodontal tissue attachment levels. Treatment effectiveness and tooth survivability with time are dependent on effective initial therapy but the initial prognosis and patient compliance both impact tooth survival. The compliance of periodontal patients can generally be categorized as: Non-compliance when the patient doesn’t comply at all; Erratic compliance when patients comply occasionally; Complete compliance when the patient complies 75% of the time (Wilson, 1996). Compliance involves active participation with prescribed oral hygiene regimen and compliance with suggested maintenance schedules. Both are critically important for maintaining periodontal health.

The prognosis assigned to a dentition is a prediction of the probable course of the disease and its effect on that dentition. Assigning a prognosis to a particular tooth is possibly the most important step in our clinical examination since the periodontist will base most of his treatment decisions on the prognosis.
II. 4. i Long-term maintenance

**Definition of Long-term Maintenance**

Due to the short-term positive results of scaling and root planing (S/RP) alone, it has been advocated that any long-term study that wants to test the true effectiveness of maintenance (after the short-term results of curettage), should exceed 3 years of observation after initial treatment (Ramfjord et al., 1973). Most longitudinal studies nowadays follow-up treated patients for at least 5 years if not more (Ramfjord, 1973; Pihlstrom, 1983; Konig, 2002) but some have extended their studies to 19-22 years (Hirschfeld & Wasserman, 1978; McFall, 1982)

**Stability of Shallow and Deep Pockets**

Different methods of treatment have been examined over the years including scaling and root planing (S/RP), modified widman flap (MWF), pocket elimination or reduction surgery etc. S/RP alone seems to have a great result in pocket depth reduction initially (reduction of 3.85 mm after 1 year) in deep pockets but these pockets gradually return to near baseline values within 6 years (Ramfjord et al., 1968 & 1973). Knowles et al (1979) showed similar results where pocket depths over 4 mm would gradually return to initial readings over time when only S/RP was performed. However, when a surgical approach was adopted the results were different. Surgery created a significantly greater probing depth reduction and the greater the initial pocket depth the greater its resolution.
(Morrison et al., 1980; Knowles et al., 1979). After 6.5 years of maintenance in the ≥7 mm pockets there was a reduction of <1.5 mm in the S/RP teeth while the Flap teeth had a reduction of 2.12 mm (Pihlstrom et al., 1983). There was a pocket depth resolution of 4-5 mm when the initial depth was over 7 mm, which was well sustained over 8 years of maintenance (Knowles et al., 1979). In a study of similar length, the average probing depth changed from 7.7 to 3.4 mm (4.3 mm of improvement) after initial therapy and surgery and to 4.4 after the 8-year maintenance period (Konig et al., 2002). In this same study, the percentage of teeth with a deep pocket (>6 mm) decreased from 28% to 0.1% after completed periodontal therapy. At the 8-year maintenance mark this number remained very low, i.e. 2.7%.

**Treatment Modality and Long-Term Stability**

When it comes to loss of attachment there is a great difference between surgical periodontal therapy and conservative periodontal treatment, i.e. S/RP. There is a significant loss of attachment initially from surgery vs. S/RP but with time these differences become no longer significant (-2.62 mm vs. -2.31 mm respectively after 7 years) (Ramfjord et al., 1973). In the very deep pockets (7-12 mm) surgery provides a significant gain of attachment (as does S/RP) and this gain is partially maintained at 8 years, which is not the case for subgingival curettage (Knowles et al., 1979). Maxillary bicuspid and molars seem to show the least significant gain in attachment after 8 years although the differences between tooth types was not significant (Ramfjord et al., 1980). Changes in attachment levels show that at 6.5 years there are gains of 1.68 mm (for teeth
treated with S/RP) to 1.32 mm (for teeth treated with flap surgery). All initial probing depths in these teeth were over 7 mm.

Attachment loss may also lead to tooth mortality not only because of compromised periodontal support but also due to increased risk of root caries. In fact in a young adult, each 1mm of buccal clinical attachment loss increases tooth mortality risk by 20% as seen in a 26 year follow up study by Hujoel et al (1999). When there is ≥3 mm of mesial attachment loss, the root mortality risk increases by 270% (relative risk of 3.7).

**Tooth Loss**

The main goal of periodontal treatment as mentioned by Hirschfeld & Wasserman (1978) is the retention of as many teeth as possible in health, function, and comfort. Studies on tooth loss in periodontal patients provide invaluable information on various prognostic indicators and also demonstrate the long-term effectiveness of periodontal treatment and long-term maintenance. There are a few longitudinal studies investigating tooth loss during maintenance and the survivability of different teeth.

One of the classical long-term maintenance studies is the Hirschfeld & Wasserman study in 1978. This study established the symmetry of tooth loss on the right and left sides of the mouth. It is a retrospective study of 600 patients who had been in the maintenance phase of treatment for a minimum of 15 years. Nearly 60% of the patients in this study were maintained for over 20 years and 83.2% of the patients lost up to 3 teeth. In
agreement with this finding, McFall (1982) similarly found that 77% of patients lost up to 3 teeth and Nabers et al. (1988) found that 60% of patients followed up for an average of 13 years lost up to 2 teeth. However, tooth loss increased from 2% to 4% to 7% when maintenance was extended from 5.5-6.7 to 10 years (Konig et al., 2002; Checci et al., 2002; Tonetti et al., 2000). In longer term studies this number increased further to 9.8% and 10.7% when maintained for 19 and 22 years respectively (McFall, 1982; Hirschfeld & Wasserman, 1978).

RANKING OF TOOTH LOSS

The most resistant teeth to disease and eventual loss (when treated and maintained) are the canines (especially mandibular), while the most likely to be lost are the maxillary second and first molars (Hirschfeld & Wasserman, 1978; McFall, 1982). Even in patients who elected not to undergo maintenance therapy after their initial treatment with the maxillary 2\textsuperscript{nd} molars showing the highest mortality (15.6% lost) and the mandibular canines showing the lowest, i.e. 1.2% (Becker et al., 1984). The results by Hirschfeld & Wasserman (1978) and McFall (1982) were supported by a recent study by Checci et al (2002) who followed patients for an average of 6.7 years of maintenance. Becker et al. (1979) found the mandibular 2\textsuperscript{nd} molars to be most frequently lost and then followed by the maxillary molars and second premolars in periodontal patients that received no treatment in this 10-year retrospective study. The teeth most resistant to loss after 10 years of untreated periodontal disease were the canines. Konig et al. (2002) found the maxillary 1\textsuperscript{st} molars to be the most frequently extracted tooth after a minimum of 10 years of maintenance. He also showed that the likelihood of loosing a maxillary tooth
from periodontal disease in periodontally involved patients is significantly higher than loosing a mandibular tooth (10.3 vs. 6.6%).

MOLAR VS. NONMOLAR TOOTH SURVIVABILITY

When examining the response to periodontal treatment via changes in probing depth and attachment loss it seems to be only marginally related to tooth type (when moderate and deep pockets are considered). There is only a trend (not statistically significant) for less favorable results (and maintenance of those results) in the maxillary molar teeth (Ramfjord et al., 1980). When examining tooth loss however, a more recent study by Konig et al (2002) revealed significant differences in tooth loss between molars and nonmolars. Overall 17.3% of molars were extracted vs. 6% of nonmolar teeth. In the 8-year maintenance phase, 7.2% of molars vs. 2% of nonmolars were lost. Konig (2002) found 94% of non-molars surviving after a minimum of a decade in maintenance. The tooth loss reported by Hirschfeld & Wasserman (1978) and McFall (1982) was 14.6%-17% of molar teeth being lost and 3.2%-6.7% of nonmolar tooth loss after an average of 19-22 years of maintenance.

Nearly 60% of all extracted teeth during maintenance are molars (Hirschfeld & Wasserman, 1978). The high incidence of molar tooth loss may be explained by the presence of the furcation associated with multirooted teeth. The percentage of maxillary molar tooth loss increases from 17.7-20.9% (of all molars) to 33-56% when only the furcated molars are taken into account. Similar patterns are observed in the mandible where 11.5-14.6% of all mandibular molars are lost during maintenance. This number
increases to 29.5-58% when considering only the furcated molars (Hirschfeld & Wasserman, 1978; McFall, 1982). Overall molar tooth loss doubles when there is a furcation involvement. Pearlman (1993) showed a loss of 26.5% of furcated teeth but the maintenance period in his study was approximately half (10 years). All patients in these studies had received complete initial therapy (including surgery when necessary). Ross and Thompson (1978) lost 12% of maxillary molars with initial furcation invasion for an average of over 10 years. In their study they maintained these furcated maxillary molars without osseous surgery but rather with regular SPT and occlusal adjustments.

II.4.ii Patient Compliance

Compliance with Oral Hygiene Regimen

It has been shown that whenever someone ceases his oral home care, bacterial plaque will adhere to his dentition and result in the clinical signs of gingivitis. These signs are reversible when oral hygiene measures are re-established (Löe et al., 1965). Even when a strict 3-month maintenance protocol is established in periodontal patients those with poorer oral hygiene tend to exhibit significantly greater initial attachment loss from those with good oral hygiene (Ramfjord, 1982).

Most commonly therapists instruct periodontal patients to use interproximal cleaning aids (i.e. interdental brushes, dental floss, rubber tip stimulator). However, even when re-instructed regularly, fewer than 50% of patients still use these agents after 3 years.
(Johansson, 1984). In an attempt to influence patients to maintain their compliance at the initially high levels of oral hygiene regimens different measures have been proposed such as regular positive feedback and use of a disclosing agent, which seemed to be helpful in lowering plaque and bleeding scores (Glavind et al., 1983; Godin, 1976).

Compliance with Maintenance Schedule

In a study by Wilson et al (1984), where 961 patients were followed up for over 8 years it was observed that 16% complied with prescribed maintenance schedules, 49% complied erratically and 35% never returned for maintenance. When investigating tooth loss after 5 years in a subsample of 162 maintenance patients between the “complete compliance” group (seen every 3 months) vs. the “erratic compliance” group, there were no teeth lost in the first group while 22 teeth were lost in the later. Checchi et al (2002) maintained 92 patients for an average of 6.7 years. Eighty percent (80%) of the compliant patients who presented in the clinician’s office for maintenance every 3-4 months lost no teeth. Twenty percent (20%) of patients lost 1-3 teeth while no patients lost over 3 teeth. Erratically compliant patients do less well during maintenance. Over 40% of them lost teeth with 10% losing more than three. The likelihood of the erratically compliant patients to lose teeth after completion of initial therapy is 5.6 times higher.

It is important to acknowledge that even with less than ideal oral hygiene, if patients are maintained on a regular maintenance schedule (every 3 months), pocket depths and attachment levels after active therapy may be maintained at similar levels up to 7 years (Ramfjord et al., 1982).
Factors Associated with Prognosis

McGuire and Nunn (1991, 1996a, 1996b, 1999) have extensively studied the association between the assignment of a prognosis on a periodontally involved tooth and the survivability of that tooth. One should keep in mind though that they would assign a prognosis after the initial treatment was completed and just before maintenance phase was started. According to their studies, factors taken in consideration into assigning a prognosis were the following: percentage, distribution and type of bone loss, probing depth, presence and severity of furcations, mobility, crown-to-root ratio, root form, pulpal involvement, caries, strategic value. Other factors may play a significant role in the overall prognosis such as age, medical status, rate of progression, patient cooperation, etc. Environmental factors also play an important role that cannot be ignored. Smoking and diabetes both double the likelihood of a tooth being re-assigned a poorer prognosis (at 5 years) that its original prognosis (McGuire & Nunn, 1996).

Prognosis Categories

In most studies there are 5 different classes of prognoses; good, fair, poor, questionable, hopeless. In the Faculty of Dentistry at The University of British Columbia, the questionable group is not used. Instead the most severely involved questionable teeth are
usually incorporated into the hopeless group while the less severely involved are part of the poor prognosis category, leaving 4 different prognosis classes.

HOPELESS

In general most clinicians agree on the “hopeless” prognosis as one being when the tooth cannot be maintained in health, comfort and function. A hopeless prognosis was accompanied by a recommendation of extraction (Becker et al., 1984; McGuire, 1991). Many times patients elect not to extract hopeless teeth. Sixty two percent (62%) of hopeless teeth that are not extracted in the initial phase of treatment, end up being extracted within an average of 5.8 years (McGuire & Nunn, 1996). It is surprising that only 33-38% of teeth assigned a prognosis of “hopeless” are actually extracted during initial treatment (Becker et al., 1984; McGuire et al., 1996).

QUESTIONABLE

Teeth exhibiting more than 50% attachment loss and class II or class III furcations are common characteristics of these teeth. A higher mobility grade of “2” is also very common. During the maintenance phase of treatment (5.2-5.5 years), the questionable teeth that are lost may vary; 37.2% during the entire treatment (Becker et al. 1984) and 26.6% during maintenance alone (McGuire & Nunn, 1996).

POOR

Teeth were given such a prognosis when there was up to 50% attachment loss with class I or class II furcation involvements to maintain. Mobility also played a significant
role (McGuire, 1991). Wilson (1987) showed that when a tooth is assigned a poor prognosis, periodontal complications were by far the most common reason for its extraction but this did not apply to the fair and good category. The poorer a prognosis given to a tooth the shorter its average survival time. For example survivability of poor teeth is actually half of that found in fair teeth (Wilson et al., 1987; McGuire & Nunn, 1996).

FAIR
Teeth given a fair prognosis usually have up to 25% attachment loss and/or class I furcation involvements. However, these teeth (with a furcation involvement or not) can be well maintained when the patient is compliant and diligent with oral hygiene (McGuire, 1991). Only 7.8% of fair teeth were extracted in a maintenance period of 10 years (McGuire & Nunn, 1996).

GOOD
When given a good prognosis the tooth provides adequate periodontal support and is easy to maintain by the patient and the clinician. Becker et al (1984) showed a total loss of 3% of initially good teeth. McGuire (1996) lost 2% of good teeth during maintenance. In most studies it seems that the greater majority of teeth are given a good prognosis in patients with moderate to severe chronic periodontitis (Wilson et al., 1987; Becker et al., 1984; McGuire, 1991; McGuire & Nunn, 1996). Interestingly, Wilson et al (1987) found that of the good teeth that are lost, only 29% of them are lost due to eventual periodontal complications, while 71% of them are lost due to other reasons.
Conclusion

Assigning an appropriate prognosis may prove to be very difficult. Many clinicians will give more weight to certain clinical parameters than others. It is important that the survival rates are consistent with prognosis assigned to the teeth during the initial exam. The prognosticated teeth should differ significantly on their survivability depending on which prognosis category they belong to. Although scaling and root planing alone has been proven to be sufficient for long-term maintenance in shallower pockets, most studies advocate a surgical approach during initial treatment when treating deeper pockets. This surgical preference may seem to be indicated even more so in molar teeth where tooth loss is generally increased. Results from different maintenance studies tend to suggest that frequent recalls and appropriate quality of home care can have a dramatic impact on attachment levels and eventually tooth loss.
Chapter Three  Rationale

The aim of all periodontal treatment is to first improve the clinical parameters associated with disease and second to maintain periodontal attachment levels with time and ultimately minimize the number of teeth that are lost. Naturally, we expect to provide a periodontium that is maintainable for the patient. Therefore, any assessment of a clinical treatment program should approach the evaluation from each of these perspectives. In addition, if sufficient patients are present in test groups we will examine whether ethnic, medical diseases and smoking impact periodontal disease stability. Since patients presenting for treatment are ultimately more interested in maintaining these teeth it would be of great help to be able to forecast the frequency of tooth loss based on the initial individual tooth prognosis assigned.

Aims

1. To examine the population characteristics of periodontal patients with moderate-severe periodontal disease that are in our patient recall pool, especially in relation to their medical status and ethnicity.

2. To evaluate the effectiveness of the treatment and maintenance program at the Graduate Periodontics clinic at The University of British Columbia. Specifically investigate the stability of probing depths, attachment levels and tooth loss.

3. To assess the impact that initial probing depth measurements (over or under 7mm) and tooth type (molar or nonmolar) have on the effectiveness of our treatment.
4. To determine the average duration of maintenance and frequency of recall visits and the effect they may have on the stability of attachment levels.

5. To examine if “known” risk factors such as smoking and disease severity have a significant impact on the treatment outcome (in means of attachment level changes) of our patient population.

6. To investigate the long term survivability of prognosticated teeth in relation to the individual prognosis assigned to them during the initial new patient exam in our group of actively participating maintenance patients (regular SPT program).
Chapter Four  Materials and Methods

IV.1 Information on the Graduate Periodontics Program at the University of British Columbia

Introduction

All the information acquired for this project is entirely derived from Graduate Periodontics clinic charts at The University of British Columbia, Vancouver, Canada. The treatment offered in this clinic is delivered by the graduate periodontics residents and/or professional hygienists that are hired by the graduate clinic to participate in the delivery of maintenance care to the patients. All graduate residents participate in the initial therapy of their patients and are involved in their maintenance. Patients are recalled based on their needs and all are re-examined annually. The measurements from these annual re-charting appointments are then assessed and compared to readings from previous years and treatment needs based on changes are planned.

Patient Pool

The patients are received from 3 different sources; the undergraduate dental clinic, referrals from private practice or walk-in patients by appointment. Patients examined in the undergraduate clinic and deemed to have advanced needs are referred to the graduate periodontics clinic. Some patients may proceed to receive their initial therapy by the
undergraduate student that has been assigned to them but at re-evaluation they are referred to the graduate clinic for additional therapy. When patients are referred for their periodontal treatment by general practitioners, the patients are screened by either the graduate students or by periodontal instructors. In most cases the patient will proceed to the graduate clinic and then be assigned to a graduate resident who will initiate their treatment. Some patients who don’t have a regular dentist may make a screening appointment where again it will be decided if the patient will proceed to the graduate or undergraduate clinic.

Graduate Residents and Hygienists

The residents are all dentists who have graduated from an accredited dental school. They are trained to use the same classification systems for the data collection, data interpretation and diagnosis. The data collection is done at the initial exam using a standardized questionnaire and clinical examination package. Once the residents complete the comprehensive patient exam they will formulate a diagnosis, prognosis and treatment plan.

A registered dental hygienist who works part time at the graduate clinic provides treatment during the maintenance phase of the patients’ treatment. However, students still remains actively involved in the maintenance scaling and root planning delivered to his/her patients.
IV.2 Therapy Performed in Study Sample

All patients in our study population received the appropriate active treatment (nonsurgical and surgical therapy) prior to entering a maintenance program. This active phase included oral hygiene instructions, initial scaling and root planing, re-evaluation (6-8 weeks later) and surgeries or extractions that are deemed necessary. A debridement schedule (usually 3-4 months) of regular SPT appointments was assigned for the first year of treatment. At this time during the annual recall exam (where re-charting takes place) the initial phase of therapy is evaluated and patients enter their maintenance programs (phase III).

IV.3 Collection and interpretation of data

Entry Criteria

All data were collected from the charts of those patients who were on maintenance in the Graduate Clinic of Periodontics at UBC. The patients selected were all seen within the last 6 years (1997) when an electronic entry system was introduced to the graduate periodontics clinic. 118 patients were on regular maintenance at some time since 1997. There were 3 major inclusion criteria for a patient’s chart to be entered in this study.

- The patient had to be at least 17 years old at the initial exam date.
- The patient had to have a minimum of 16 permanent teeth present at the initial exam.
- The patient had to be in maintenance for a minimum of 24 months.

After all the above restrictions, the charts of 100 patients were used for this study.

Compilation of Collected data

All data were collected by the same examiner (Dr. Irinakis) who was solely responsible for assigning a diagnosis and prognosis based on the chart data and the radiographic survey. When extracting the data, the examiner formulated a “data collection sheet” which contained all the information for each patient on an excel sheet (Appendix No1). In the end a complete excel workbook was fabricated that contained all 100 “data collection sheets” for every patient.

The data collected included the following:

1. Patient’s name was replaced by a 3-digit code
2. Date of birth
3. Date of initial examination when the patient first came to the clinic and had his/her clinical charting completed
4. Clinical charting information from 3 different dates were collected:
   1st reading→ data from the initial examination date
   2nd reading→ data collected after initial treatment was completed (without necessarily including all the surgical procedures that had been planned) within the following 12 months (on average)
   3rd reading→ Most recent date the data were collected. This was the last clinical evaluation associated with maintenance.
5. Mobility

The Miller classification (1985) was used in a slightly modified manner as it is used at the UBC dental clinic:

Class I → Any horizontal mobility more than normal and up to 1mm
Class II → Any horizontal mobility between 1-2mm
Class III → Any horizontal mobility over 2mm and/or mobility in the vertical direction

Wherever the graduate student has a plus (+) or a half (1/2) in his/her classification the immediately lesser classification was assigned. If there was no mobility (beyond physiologic) present a zero (0) was assigned.

A numeric reading will be used on the Data Collection sheet.

“0” mobility → number 0
Class I mobility → number 1
Class II mobility → number 2
Class III mobility → number 3

6. The permanent teeth that were examined in this study were numbered according to the FDI numeration

7. In the column where the probing depth (in mm) is given (PD), the deepest of the 6 readings on each tooth was used.

8. Recession was recorded in mm from the CEJ. If there is a facial or lingual restoration extending beyond the CEJ or covering it, the apical margin of that restoration is used to determine the recession value for that tooth. The deepest recession was recorded for that tooth. If there was no recession a zero (0mm) was
assigned for that tooth.

9. When assigning a furcation class to a tooth, the most severe furcation involvement was recorded.

According to Hamp’s Classification:

Class I → Furcation involvement less than 3mm
Class II → Furcation involvement more than 3mm but not through-and-through
Class III → Furcation involvement is through-and-through

When measuring the furcation involvement in Hamp’s classification we measure the horizontal attachment loss in that furcation from a tangent line that connects the outermost convexities of the roots forming that particular furcation. If there was no clinically detectable furcation involvement a zero (0) was assigned.

A numeric value similar to the Mobility section was assigned for each classification in the Data Collection sheet.

10. Prognosis was assigned using the following guidelines that the graduate periodontics students follow when formulating treatment plans for the UBC patients. These guidelines include, but are not limited to, the prognosis categories suggested by McGuire MK (1991) (Table 1).
### Table No.1
**Prognosis Guidelines**

| Good Prognosis: | (one or more of the following) adequate periodontal support and control of the etiologic factors to assure the tooth would be relatively easy to maintain, assuming proper maintenance. |
| Fair Prognosis: | (one or more of the following) attachment loss to the point that the tooth could not be considered to have a good prognosis and/or Class I furcation involvement. The location and depth of the furcation would allow proper maintenance with good patient compliance. |
| Poor Prognosis: | (one or more of the following) moderate attachment loss with Class I and/or Class II furcations. The location and depth of the furcations would allow proper maintenance, but with difficulty. |
| Questionable Prognosis: | (one or more of the following) severe attachment loss resulting in a poor crown-to-root ratio. Poor root form. Class II furcations not easily accessible to maintenance care or Class III furcations. 2+ mobility or greater. Significant root proximity. |
| NOTE: | This category was not used and the following change took place: The prognosis of “questionable” was replaced with “poor” for the purposes of this study. |
| Hopeless Prognosis: | Inadequate attachment to maintain the tooth in health, comfort, and function. Extraction was performed or suggested. |

Table 1. Guidelines in assigning a prognosis to a tooth as stated by McGuire and Nunn (1991).

Additional criteria used to determine prognosis includes:

- Age, medical status, percentage/type/distribution of bone loss, caries, oral habits, occlusal factors.
Each prognosis was given a numeric value:

- Hopeless → Numeric value “1”
- Poor → Numeric value “2”
- Fair → Numeric value “3”
- Good → Numeric value “4”

11. The type and number of surgeries were numerically coded.

- 0 → No surgery
- 1 → Pocket reduction surgery or OFD/Osteoplasty or Tunneling/Osseous
- 2 → Regeneration with or without bone grafting
- 3 → FGG (free gingival graft)
- 4 → CTG (connective tissue graft)

The overall number of periodontal surgeries will also be mentioned for each patient.

12. The number of SPT visits was assigned to each patient along with the time period within which these visits took place.

13. Attachment loss from the CEJ to the bottom of the probeable pocket was determined. When there was no recession present the attachment loss was calculated as the PD - 2mm (average depth of a healthy sulcus). The value was given in millimeters.

14. The teeth present at each separate data collection date were mentioned.

15. Significant medical concerns were recorded. A numerical value was given to the following positive medical findings:

- 0 → no medical concerns
- 1 → Diabetes type I
- 2 → Diabetes type II
3→ Cardiovascular disease (stroke, heart attach, angina pectoris)

4→ High blood pressure

5→ Osteoporosis

16. Smoking was recorded for in 3 different categories

   Current smokers: Numeric value of “1”

   Previous smokers (quit within 5 years prior to initial exam): Numeric value of “2”

   Non-smokers (Patients that have never been smokers or quit > 5y from date of initial exam): Numeric value of “3”

17. The initial severity of the periodontal disease was recorded. The severity of the disease will be categorized based on the classification by Wasserman & Hirschfeld (1978) and McFall (1982) slightly modified in the Moderate disease category:

   Mild: Pockets of 4mm or less, generally with gingival inflammation and subgingival calculus deposits

   Moderate: Pockets of 4 to 7 mm present about a number of teeth. (Addition: only one tooth could have a PD over 7mm as long as there were no furcations in other areas ≥ Class 2).

   Severe: Pockets deeper than 7mm, furcation involvement of at least one tooth.
IV.4 Statistical Analysis

Observational analyses including descriptive and inferential statistics were used with SPSS software (SPSS Inc.). Descriptive analyses summarized and organized the collected data. The mean values, histograms and relative frequency tables were tabulated. In addition, ranges, standard deviations and standard errors were calculated and labeled on the bar graphs.

Inferential analyses allowed for association inferences using correlation, independent t-test and ANOVA. Pearson’s correlation examined the magnitude and the direction of the linear relationship between variables. Firstly, the independent t-test was used to evaluate the differences in means between two groups. ANOVA, then tested for significant differences between the means of the categorical and numerical variables.
Chapter V Results

V.1 Study Population characteristics

100 periodontal patients who had been treated in the graduate periodontics clinic at the University of British Columbia (UBC) and maintained for a minimum of 2 years were studied. The majority of the patients were Caucasian (74%) and the rest were Asian (14%), Indo-Canadian (6%) and of Middle Eastern origin (6%). Due to the overwhelming majority being Caucasian it was decided not to study the population according to different ethnic groups but rather as a whole.

Overall there were 69% female and 31% male patients and all patients had either moderate or severe periodontal disease with equally distributed percentages of males and females in both disease categories (Table 2). None of the patients in the UBC clinic had mild periodontal disease. Notably, 36% were initially diagnosed as having moderate periodontal disease while 64% had severe disease (Table 3).

The clinical measurements taken from all teeth but the 3rd molars were examined. The number of teeth per patient present at the initial exam ranged from 16-28 with an average of 24.9 ±2.9 (mean ± SD) teeth present at the initial exam. When the initial treatment was completed (prior to entering the maintenance period), a re-evaluation was performed during which it was noted that 50% of the patients had relatively complete dentitions, i.e. ≥25 teeth (McFall, 1982) (Table 4). On the other hand there were 50% of the study
sample that had lost at least 4 teeth (other than 3rd molars) prior to and after initial therapy.

### TABLE No. 2

<table>
<thead>
<tr>
<th>Characteristics of the Patient Population (n = 100)</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Overall Patients</strong></td>
<td></td>
</tr>
<tr>
<td>Males (n &amp; %)</td>
<td>31 (31%)</td>
</tr>
<tr>
<td>Females (n &amp; %)</td>
<td>69 (69%)</td>
</tr>
<tr>
<td><strong>Moderate Periodontal Disease</strong></td>
<td></td>
</tr>
<tr>
<td>Males (n &amp; %)</td>
<td>11/36 (30.5%)</td>
</tr>
<tr>
<td>Females (n &amp; %)</td>
<td>25/36 (69.5%)</td>
</tr>
<tr>
<td><strong>Severe Periodontal Disease</strong></td>
<td></td>
</tr>
<tr>
<td>Males (n &amp; %)</td>
<td>20/64 (31.25%)</td>
</tr>
<tr>
<td>Females (n &amp; %)</td>
<td>44/64 (68.75%)</td>
</tr>
</tbody>
</table>

Table 2. Gender description of our study population and its distribution according to disease severity

### TABLE No. 3

<table>
<thead>
<tr>
<th>Periodontal Disease Severity of the Patient Population-1 (n = 100)</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with Mild Disease (n &amp; %)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Patients with Moderate Disease (n &amp; %)</td>
<td>36 (36%)</td>
</tr>
<tr>
<td>Patients with Severe Disease (n &amp; %)</td>
<td>64 (64%)</td>
</tr>
</tbody>
</table>

*For definitions see Materials & Methods*

Table 3. Distribution of periodontal disease amongst the members of our population sample. Notably, only moderate and severe periodontitis patients are present.
TABLE No. 4

Teeth Present after Initial Therapy

<table>
<thead>
<tr>
<th>Number of teeth present</th>
<th>Number of patients</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥25</td>
<td>50</td>
<td>50%</td>
</tr>
<tr>
<td>21-24</td>
<td>34</td>
<td>34%</td>
</tr>
<tr>
<td>16-20</td>
<td>15</td>
<td>15%</td>
</tr>
<tr>
<td>11-15</td>
<td>1</td>
<td>1%</td>
</tr>
<tr>
<td>6-10</td>
<td>0</td>
<td>0%</td>
</tr>
</tbody>
</table>

Table 4. Distribution of patients according to the number of teeth that were present before entering the maintenance phase of their treatment

The average age of the patients at the initial exam was 49.58 ± 11.87 years (mean ± SD) (Table 5). Seventy-eight (78%) patients were over 40 years old, while 21% were at least 60 years of age. After an average of nearly a year of initial therapy (11.4 ± 5.1 months), the patient entered maintenance where they remained there for an average of 5.22 years (Table 6). However, 19 patients were in maintenance for at least 8 years, while 11 of them were in maintenance for at least 10 years. On the other hand, half of the patients (50%) were in maintenance for less than 4 years. Based on the duration in maintenance patients were also grouped as short-term with 2-5 years and long-term with a minimum of 5 years of maintenance. Sixty-one patients (61%) were short-term (ST), while 39% were long-term (LT).
**TABLE No. 5**

**Distribution of Sample by Age at Initial Treatment**

<table>
<thead>
<tr>
<th>Patient's age</th>
<th>Number of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>17-29 (&lt;30)</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>30-39</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>40-49</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>50-59</td>
<td>32</td>
<td>32</td>
</tr>
<tr>
<td>60-76 (≥60)</td>
<td>21</td>
<td>21</td>
</tr>
</tbody>
</table>

Table 5. Distribution of patient population according to their age at the time of their initial exam.

**TABLE No. 6**

**Distribution of Sample by Years of Maintenance**

<table>
<thead>
<tr>
<th>Years of Maintenance</th>
<th>Number of patients</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-3</td>
<td>31</td>
<td>31</td>
</tr>
<tr>
<td>3-4</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>4-5</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>5-6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>6-7</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>7-8</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>8-9</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>9-10</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>≥ 10</td>
<td>11</td>
<td>11</td>
</tr>
</tbody>
</table>

| Short Term: 2-5   | 61                 | 61      |
| Long Term: ≥ 5   | 39                 | 39      |
| Overall Average: 5.22 | 100               | 100     |

Table 6. The patients are divided into different groups based on how many years they have been in maintenance. Additional grouping has been conducted and the patients have been assigned to either a short-term or a long-term maintenance group.

We reviewed the medical history of our patient for systemic conditions that may impact progression of periodontal diseases. We found that 21% of the patients had at least one of the systemic conditions investigated (Table 7). However, only 5% had diabetes mellitus, osteoporosis or cardiovascular disease, and only 14% had elevated blood pressure at the
time of initial exam. Due to these small numbers, the systemic conditions present and their relationship/impact was not explored further.

The ratio of nonsmokers vs. smokers is 2.27:1 with smokers representing 29% of the population while five patients (5%) were previous smokers. An average of $2.22 \pm 2.0$ (mean ± SD) surgeries/patient were performed on this population. However, when concentrating on the 76% of patients who had a surgery this average number (for this subgroup) increases to $2.91 \pm 1.7$. This means that the surgery patients had nearly 3 surgeries each during the study period (initial therapy & maintenance).

**TABLE No. 7**

<table>
<thead>
<tr>
<th>Summary of Medical Findings and Habit History</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients having any of the below disorders</td>
<td>21 (21%)</td>
</tr>
<tr>
<td>Diabetes (both types) (n &amp; %)</td>
<td>5 (5%)</td>
</tr>
<tr>
<td>↑BP (n &amp; %)</td>
<td>14 (14%)</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Cardiovascular Disease (n &amp; %)</td>
<td>4 (4%)</td>
</tr>
</tbody>
</table>

* Some patients positively reported more than one of the above systemic conditions

| Patients receiving surgical therapy | 76 (76%) |
|------------------------------------|         |
| Average surgeries performed        | $2.22 \pm 2.29$ |
| Number of surgeries on the 76 surgical patients | $2.91 \pm 1.77$ |

| Current smokers (n & %) | 29 (29%) |
| Previous smokers (n & %) | 5 (5%)   |
| Non-Smokers (n & %)     | 66 (66%) |

Table 7. Three aspects of the patients are outlined in this table. Medical conditions that we investigated, information on periodontal surgeries and on smoking status.
V.2 Change in clinical parameters during initial therapy and maintenance

V.2.i Change in pocket depth & attachment loss over the study period

Mean probing depths decreased from initial visit through maintenance (Figure 1). The attachment levels improved initially and slightly deteriorated during maintenance. Changes in probing depth and attachment level were categorized into deep and moderate pockets for further assessment (Figure 2). In deeper pockets (≥ 7 mm) and moderate pockets (4-6 mm) of the teeth that survived (336 severely vs. 1345 moderately involved teeth) the entire treatment period, similar changes were noticed. In the deep pockets the initial average probing depth of 7.7 mm decreased to 5.6 mm (p<0.01) at the end of initial therapy and finally to 4.9 mm (change not significant). The attachment levels changed from 7.8 mm to 6.3 mm (p<0.01) to 6.6 mm respectively (change not significant). Moderate pockets decreased from 4.9 to 4.2 to 4.0 mm (change not significant). The attachment loss in these teeth changed from 4.9 to 4.6 to 4.7 mm in the final exam (change not significant). Although both moderate and deep pockets showed similar trends of disease progression/remission, the deeper pockets showed significantly better improvement in probing depth in all different examinations (p<0.01). The deeper pockets also showed significantly better improvement in attachment levels during the initial therapy and the entire treatment (active therapy + maintenance) (p<0.01) but not during maintenance.
Subsequently, individual groups of teeth were examined. During the maintenance period, all teeth (2425 teeth present at beginning of maintenance period) showed relatively stable pocket depths and in all groups the pocket depths continued to decrease except of the Mx 2\textsuperscript{nd} premolars that increased by 0.02 mm (Table 8). Generally, the deeper the initial probing depth readings were the greater those pockets resolved. The apparent decrease in pocket depth may occur from either gain in attachment or increase in recession. The molar teeth that survived (not including extracted teeth) showed decreases in pocket depth from 6.2 to 5.1 mm during the entire treatment (p<0.05) (Figure 3). The nonmolar teeth showed similar improvement from 5.2 to 3.9 mm (p<0.01).

The attachment levels of all teeth remained clinically stable as well. Although most teeth showed some level of continuing attachment loss during the maintenance phase, the range of this attachment loss was from 0.03-0.40 mm (Table 9). Four groups of teeth actually showed attachment gain during maintenance that ranged from 0.07 mm (Md central incisors & Mx 1\textsuperscript{st} premolars) to 0.46 mm in Mx canines and Mx 2\textsuperscript{nd} molars. It becomes apparent that the probing pocket depth decrease during maintenance was not accompanied by a general attachment gain. This would indicate that most of the pocket depth resolution was attributed to concurrent recession rather than attachment gain. The nonmolar teeth showed an overall improvement in attachment levels over the entire treatment (5.20 to 4.65 to 4.60 mm). This differed significantly from the picture that the molars showed (p<0.01). The final attachment levels of the molar teeth were worse than the initial attachment levels i.e.6.3 to 5.9 to 6.6 mm (Figure 3).
Molars and nonmolars showed differences in their response to treatment in both the moderate pockets and the deeper pockets (Table 10). In the moderate probing depth group, both tooth types had a sustained overall decrease in probing depth after 6 years of treatment (1 year of active therapy + 5 years of maintenance). The molars decreased their PD by 0.43 mm and the nonmolars by 0.92 mm. However, the molars had an overall attachment loss of 0.59 mm after 5 years in maintenance while the nonmolars continued to show sustained gain of 0.37 mm. When viewing the data in the deeper pockets where usually our aim of treatment will be focused, the molars showed an overall improvement of 2.26 mm and the nonmolars of 3.22 mm. Both groups of teeth also exhibited a sustained gain in attachment of 0.40 and 2.20 mm respectively.

Figure No. 1

Mean Probing Depths & Attachment Levels

Figure No. 2

Figure 1. The overall probing depths and attachment levels at the 3 different times of examination are plotted on this chart. All values are averages.
Change in Clinical Parameters during Tx of Surviving Teeth; Deep vs. Moderate Pockets

Figure 2. The positive and negative average changes of attachment levels and probing depths are represented. In dark ink are the teeth that had at least a 7 mm PD reading and in light ink are the teeth that had a PD reading of 4-6 mm at the time of the initial exam.

TABLE No. 8

Relative Change of Probing Depth during Treatment & Maintenance

<table>
<thead>
<tr>
<th>Groups/ FDI number</th>
<th>Averages (mm)</th>
<th>δ (during Maintenance)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>After Initial Tx</td>
</tr>
<tr>
<td>Mx 2nd Molars</td>
<td>5.76</td>
<td>4.88</td>
</tr>
<tr>
<td>Mx 1st Molars</td>
<td>5.39</td>
<td>4.44</td>
</tr>
<tr>
<td>Mx 2nd Premolars</td>
<td>4.68</td>
<td>3.85</td>
</tr>
<tr>
<td>Mx 1st Premolars</td>
<td>4.72</td>
<td>4.13</td>
</tr>
<tr>
<td>Mx Canines</td>
<td>4.70</td>
<td>3.91</td>
</tr>
<tr>
<td>Mx Lateral Incisors</td>
<td>4.40</td>
<td>3.57</td>
</tr>
<tr>
<td>Mx Central Incisors</td>
<td>4.35</td>
<td>3.39</td>
</tr>
<tr>
<td>Md Central Incisors</td>
<td>4.17</td>
<td>3.28</td>
</tr>
<tr>
<td>Md Lateral Incisors</td>
<td>4.05</td>
<td>3.64</td>
</tr>
<tr>
<td>Md Canines</td>
<td>4.22</td>
<td>3.70</td>
</tr>
<tr>
<td>Md 1st Premolars</td>
<td>4.24</td>
<td>3.69</td>
</tr>
<tr>
<td>Md 2nd Premolars</td>
<td>4.45</td>
<td>3.87</td>
</tr>
<tr>
<td>Md 1st Molars</td>
<td>4.96</td>
<td>4.11</td>
</tr>
<tr>
<td>Md 2nd Molars</td>
<td>5.44</td>
<td>4.42</td>
</tr>
</tbody>
</table>

Table 8. The average probing depth measurements of each individual tooth category are presented during the 3 different appointments that readings were taken, i.e. Initial Exam, Re-evaluation (after 1 year) and Final Exam.

TABLE No. 9
### Progression of Attachment Loss during Treatment

<table>
<thead>
<tr>
<th>Groups/ FDI number</th>
<th>Averages (mm)</th>
<th>δ (during Maintenance)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>After Initial Tx</td>
</tr>
<tr>
<td>Mx 2&lt;sup&gt;nd&lt;/sup&gt; Molars</td>
<td>17/27</td>
<td>5.77</td>
</tr>
<tr>
<td>Mx 1&lt;sup&gt;st&lt;/sup&gt; Molars</td>
<td>16/26</td>
<td>6.43</td>
</tr>
<tr>
<td>Mx 2&lt;sup&gt;nd&lt;/sup&gt; Premolars</td>
<td>37/47</td>
<td>4.76</td>
</tr>
<tr>
<td>Mx 1&lt;sup&gt;st&lt;/sup&gt; Premolars</td>
<td>36/46</td>
<td>4.83</td>
</tr>
<tr>
<td>Mx Canines</td>
<td>12/22</td>
<td>4.79</td>
</tr>
<tr>
<td>Mx Lateral Incisors</td>
<td>15/25</td>
<td>3.91</td>
</tr>
<tr>
<td>Mx Central Incisors</td>
<td>14/24</td>
<td>3.71</td>
</tr>
<tr>
<td>Md Central Incisors</td>
<td>11/21</td>
<td>4.91</td>
</tr>
<tr>
<td>Md Lateral Incisors</td>
<td>13/23</td>
<td>5.07</td>
</tr>
<tr>
<td>Md Canines</td>
<td>31/41</td>
<td>4.09</td>
</tr>
<tr>
<td>Md 1&lt;sup&gt;st&lt;/sup&gt; Premolars</td>
<td>33/43</td>
<td>4.20</td>
</tr>
<tr>
<td>Md 2&lt;sup&gt;nd&lt;/sup&gt; Premolars</td>
<td>32/42</td>
<td>4.46</td>
</tr>
<tr>
<td>Md 1&lt;sup&gt;st&lt;/sup&gt; Molars</td>
<td>34/44</td>
<td>3.90</td>
</tr>
<tr>
<td>Md 2&lt;sup&gt;nd&lt;/sup&gt; Molars</td>
<td>35/45</td>
<td>5.44</td>
</tr>
</tbody>
</table>

Table 9. The average attachment level measurements of each individual tooth category are presented during the 3 different appointments. The negative signs in the last column represent attachment loss. When there is no negative sign, there is attachment gain in that tooth group.

**Figure No. 3**

**Change in Clinical Parameters during Tx of Surviving Teeth; Molars vs. Nonmolars**

Figure 3. The positive and negative changes of attachment levels and probing depths of molars and nonmolars are plotted against each other. The dark ink is used to depict molar teeth while the light ink nonmolars. Interrupted lines represent attachment level changes, while continuous lines represent PDs.
TABLE No. 10

Changes in Clinical Parameters in Relation to Tooth Type and Initial Pocket Depth

<table>
<thead>
<tr>
<th></th>
<th>4-6 mm PDs</th>
<th></th>
<th>7 mm PDs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>Final</td>
<td>Initial</td>
<td>Final</td>
</tr>
<tr>
<td>Molars</td>
<td>5.21</td>
<td>4.78</td>
<td>7.91</td>
<td>7.51</td>
</tr>
<tr>
<td>NonMolars</td>
<td>4.79</td>
<td>3.87</td>
<td>7.59</td>
<td>4.37</td>
</tr>
</tbody>
</table>

Table 10. Comparison between molars and nonmolars in the improvement they present in attachment levels and pocket depths. The comparison has been divided into moderate and deep probing depths.

V.2.ii Tooth loss during maintenance phase

There is a well-established report of bilateral symmetry in periodontal disease (Miller & Seidler, 1942; Wasserman et al., 1971, 1972; Bossert & Marks, 1956; Wade, 1966). Therefore, all measurements from right and left teeth were pooled together. There were 86 teeth lost during initial treatment and 112 lost during maintenance, i.e. 4.64% of all teeth treated in maintenance were lost (Table 11). From these latter teeth, 68 were molars, which accounted for 60% of all extracted teeth in maintenance. On the other hand, 44 nonmolars were lost after initial treatment, i.e. 2.4% of the anteriors and premolars that were present at the beginning of maintenance. The likelihood of a molar tooth vs. nonmolar tooth being lost during maintenance is 5 times higher (Odds Ratio: 5.0). The most commonly lost teeth during maintenance were the Mx 2nd and Mx 1st molars. Mandibular 2nd and 1st molars were next most likely to be lost. The percentage loss of
individual teeth ranged from 17.51% of a Mx 2\textsuperscript{nd} molar to 0.57% of a Mx Central Incisor (Table 12). Notably, all mandibular teeth had a better chance of surviving when compared to the same teeth on the opposing arch with the central incisors being the only exception.

Presence of furcations and mobility influenced the time of tooth loss, i.e. during initial therapy vs. maintenance. The majority of the extracted molars (82.45%) had detectable furcation involvements. Moreover, the majority of extracted molars (73.7%) had class II or class III furcation involvements. Eighty percent (80%) of extracted molars with a class-I furcation survived initial treatment but were extracted in maintenance. This number (of extracted molars) decreases to 64% and 46% for class-II and class-III furcations respectively (Table 13). When evaluating mobility, 72% of non-mobile teeth (that were eventually lost) survived initial therapy compared to 41% of teeth with class-II or class-III mobility (Table 14a). Nearly 56% of extracted molars with class-II or class-III mobility were removed during initial treatment compared to 30% of molars with either no initial mobility or class-I mobility (Table 14b).
**TABLE No. 11**

**Percent Loss of Teeth during Maintenance**

<table>
<thead>
<tr>
<th>Groups/FDI number</th>
<th>Teeth present after Re-eval.</th>
<th>Lost during maintenance</th>
<th>Percent lost</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Teeth</td>
<td>2,413</td>
<td>112</td>
<td>4.64</td>
</tr>
<tr>
<td>Molars</td>
<td>571</td>
<td>68</td>
<td>11.91</td>
</tr>
<tr>
<td>Nonmolars</td>
<td>1,842</td>
<td>44</td>
<td>2.39</td>
</tr>
<tr>
<td>Mx 2(^{nd}) Molars</td>
<td>17/27</td>
<td>137</td>
<td>17.51</td>
</tr>
<tr>
<td>Mx 1(^{st}) Molars</td>
<td>16/26</td>
<td>146</td>
<td>13.01</td>
</tr>
<tr>
<td>Mx 2(^{nd}) Premolars</td>
<td>15/25</td>
<td>172</td>
<td>6.39</td>
</tr>
<tr>
<td>Mx 1(^{st}) Premolars</td>
<td>14/24</td>
<td>176</td>
<td>5.00</td>
</tr>
<tr>
<td>Mx Canines</td>
<td>13/23</td>
<td>195</td>
<td>2.05</td>
</tr>
<tr>
<td>Mx Lateral Incisors</td>
<td>12/22</td>
<td>186</td>
<td>1.61</td>
</tr>
<tr>
<td>Mx Central Incisors</td>
<td>11/21</td>
<td>176</td>
<td>0.57</td>
</tr>
<tr>
<td>Md Central Incisors</td>
<td>31/41</td>
<td>183</td>
<td>2.73</td>
</tr>
<tr>
<td>Md Lateral Incisors</td>
<td>32/42</td>
<td>188</td>
<td>1.60</td>
</tr>
<tr>
<td>Md Canines</td>
<td>33/43</td>
<td>199</td>
<td>2.01</td>
</tr>
<tr>
<td>Md 1(^{st}) Premolars</td>
<td>34/44</td>
<td>189</td>
<td>1.59</td>
</tr>
<tr>
<td>Md 2(^{nd}) Premolars</td>
<td>35/45</td>
<td>184</td>
<td>2.17</td>
</tr>
<tr>
<td>Md 1(^{st}) Molars</td>
<td>36/46</td>
<td>142</td>
<td>8.45</td>
</tr>
<tr>
<td>Md 2(^{nd}) Molars</td>
<td>37/47</td>
<td>147</td>
<td>9.52</td>
</tr>
</tbody>
</table>

Table 11. Tooth loss in maintenance according to each tooth type and collectively as molars and nonmolars. The extractions are presented here as raw numbers and as percentages. Each percentage corresponds to that specific tooth type, e.g. 17.51% represents the percent of Mx 2\(^{nd}\) molars lost only.

**TABLE No. 12**

**Ranking of Individual Teeth by Frequency of Loss**

<table>
<thead>
<tr>
<th>Groups</th>
<th>FDI number</th>
<th>Teeth present following initial Tx</th>
<th>Percent lost in maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mx 2(^{nd}) Molars</td>
<td>17/27</td>
<td>137</td>
<td>17.51</td>
</tr>
<tr>
<td>Mx 1(^{st}) Molars</td>
<td>16/26</td>
<td>146</td>
<td>13.01</td>
</tr>
<tr>
<td>Md 2(^{nd}) Molars</td>
<td>37/47</td>
<td>147</td>
<td>9.52</td>
</tr>
<tr>
<td>Md 1(^{st}) Molars</td>
<td>36/46</td>
<td>142</td>
<td>8.45</td>
</tr>
<tr>
<td>Mx 2(^{nd}) Premolars</td>
<td>15/25</td>
<td>172</td>
<td>6.39</td>
</tr>
<tr>
<td>Mx 1(^{st}) Premolars</td>
<td>14/24</td>
<td>176</td>
<td>2.84</td>
</tr>
<tr>
<td>Md Central Incisors</td>
<td>31/41</td>
<td>183</td>
<td>2.73</td>
</tr>
<tr>
<td>Md 2(^{nd}) Premolars</td>
<td>35/45</td>
<td>184</td>
<td>2.17</td>
</tr>
<tr>
<td>Mx Canines</td>
<td>13/23</td>
<td>195</td>
<td>2.05</td>
</tr>
<tr>
<td>Md Canines</td>
<td>33/43</td>
<td>199</td>
<td>2.01</td>
</tr>
<tr>
<td>Mx Lateral Incisors</td>
<td>12/22</td>
<td>186</td>
<td>1.61</td>
</tr>
<tr>
<td>Md Lateral Incisors</td>
<td>32/42</td>
<td>188</td>
<td>1.60</td>
</tr>
<tr>
<td>Md 1(^{st}) Incisors</td>
<td>34/44</td>
<td>189</td>
<td>1.59</td>
</tr>
<tr>
<td>Mx Central Incisors</td>
<td>11/21</td>
<td>176</td>
<td>0.57</td>
</tr>
</tbody>
</table>

Table 12. All teeth are ranked based on the mount of tooth loss they suffered. The percentage value represents tooth loss within each tooth type and not the percentage of loss when all teeth are considered.
# TABLE No. 13

**Furcation Involvement of Extracted Molars**

<table>
<thead>
<tr>
<th>Furcation Classification</th>
<th>Extracted Molars (n &amp; %)</th>
<th>Extracted molars during initial Tx (n &amp; %)</th>
<th>Extracted molars during maintenance (n &amp; %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>20 (17.5%)</td>
<td>9 (45%)</td>
<td>11 (55%)</td>
</tr>
<tr>
<td>Class-I</td>
<td>10 (8.8%)</td>
<td>2 (20%)</td>
<td>8 (80%)</td>
</tr>
<tr>
<td>Class-II</td>
<td>56 (49.1%)</td>
<td>20 (35.8%)</td>
<td>36 (64.3%)</td>
</tr>
<tr>
<td>Class-III</td>
<td>28 (24.6%)</td>
<td>15 (65.6%)</td>
<td>13 (46.4%)</td>
</tr>
</tbody>
</table>

Table 13. The 1<sup>st</sup> column represents the degree of furcation for the molars that were eventually extracted. The 2<sup>nd</sup> column represents the distribution of that total loss between the different classes, e.g. 8.8% of extracted molars were class-I. The 2<sup>nd</sup> and 3<sup>rd</sup> columns show the distribution of these extractions (immediate or in maintenance) within each furcation class and not amongst them.

# TABLE No. 14.a

**Mobility Characteristics of all Extracted Teeth (Molars & Nonmolars)**

<table>
<thead>
<tr>
<th>Mobility Classification</th>
<th>Extracted Teeth (n &amp; %)</th>
<th>Extracted Teeth during initial Tx (n &amp; %)</th>
<th>Extracted Teeth during maintenance (n &amp; %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>67 (33.8%)</td>
<td>19 (28.4%)</td>
<td>48 (71.6%)</td>
</tr>
<tr>
<td>Class-I</td>
<td>46 (23.2%)</td>
<td>17 (37.0%)</td>
<td>29 (63.0%)</td>
</tr>
<tr>
<td>Class-II</td>
<td>55 (50.9%)</td>
<td>27 (49.1%)</td>
<td>28 (51.0%)</td>
</tr>
<tr>
<td>Class-III</td>
<td>30 (15.2%)</td>
<td>23 (76.7%)</td>
<td>7 (23.3%)</td>
</tr>
</tbody>
</table>

Table 14a. The 1<sup>st</sup> and 2<sup>nd</sup> columns show the different mobility classes and the different distribution of extractions among these classes for all teeth in this study. The 2<sup>nd</sup> and 3<sup>rd</sup> columns show the distribution of these extractions (immediate or in maintenance) within each mobility class and not amongst them.

# TABLE No. 14.b

**Mobility Characteristics of Extracted Molars**

<table>
<thead>
<tr>
<th>Mobility Classification</th>
<th>Extracted Molars (n &amp; %)</th>
<th>Extracted Molars during initial Tx (n &amp; %)</th>
<th>Extracted Molars during maintenance (n &amp; %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>40 (35.1%)</td>
<td>10 (25%)</td>
<td>30 (75%)</td>
</tr>
<tr>
<td>Class-I</td>
<td>27 (23.7%)</td>
<td>10 (37%)</td>
<td>17 (63%)</td>
</tr>
<tr>
<td>Class-II</td>
<td>30 (26.3%)</td>
<td>13 (43.3%)</td>
<td>17 (56.7%)</td>
</tr>
<tr>
<td>Class-III</td>
<td>17 (14.9%)</td>
<td>13 (76.5%)</td>
<td>4 (23.5%)</td>
</tr>
</tbody>
</table>

Table 14b. The 1<sup>st</sup> and 2<sup>nd</sup> columns show the different mobility classes and the different distribution of extractions among these classes for the extracted molars only. The 2<sup>nd</sup> and 3<sup>rd</sup> columns show the distribution of these extractions (immediate or in maintenance) within each mobility class and not amongst the different degrees of mobility.
The teeth that were extracted during initial therapy when compared to those teeth that survived the initial therapy (extracted during maintenance) they had a significantly greater initial attachment loss (1.62 mm more loss, p<0.01), a deeper initial probing depth (0.53 mm deeper, p<0.01), and a worse initial prognosis (Table 15). Our patients showed an average tooth loss during maintenance of 1.12 teeth in 5.22 years. Tooth loss increased during maintenance as the duration of SPT increased. The short term patients had an average of 3 years in maintenance and an mean tooth loss of 0.75 lost teeth/patient. The long term group was in maintenance for an average of 8.5 years and lost 1.67 teeth/patient. However, when plotted per anum it was observed that the average annual tooth loss per patient decreased as the duration of maintenance increased (Figure 4). When further investigating 19 patients with an average of 11 years in maintenance this number remained stable, i.e. 1.68 teeth lost/patient.

Table 15. This table shows the difference in disease involvement (PD and AL) of teeth based on the time of their extraction, i.e. immediate or during maintenance. The prognosis row is present only to indicate that the teeth extracted during active therapy did indeed have a poorer prognosis. The lower the “number value” for prognosis, the worse the prognosis is.
Figure No. 4

Teeth lost per patient annually plotted against years in maintenance

Figure 4. This is a graph that shows how the number of extracted teeth/patient on a yearly basis according to which maintenance group they belonged to. The values are taken from subgroups of our total study population. The three subgroups were patients maintained for 2-5 years (n=61/average of 3 years), total population (n=100/average of 5 years) and patients maintained for 5 years or more (n=39/average of 8.5 years).

V.3 Supportive periodontal therapy (SPT) maintenance and modifiers of periodontal attachment levels

V.3.i SPT frequency and its effect on the studied clinical parameters

The patients from our study pool had an average of 2.58 visits per year, while their average duration of maintenance was 5.23 years (62.65 months) (Table 16). This means patients in the UBC Graduate Periodontics clinic came in every 4.6 months for SPT. All patients first received initial therapy (11.4 ± 5.08 months) prior to their entry into the maintenance phase. The long-term patients (maintained for 5 years or more) were maintained for an average of 8.58 ± 2.8 years.
Depending on maintenance frequency all patients were divided into 4 groups, i.e. very frequent, frequent, intermittent and infrequent (Table 17). Interestingly, 92% of all patients received regular SPTs every 3-6 months. Notably, 76% of the population was actually receiving treatment every 3-5 months. Only 8 patients (8%) were seen infrequently (less than twice per year). These patients were maintained for a period of over 7 years (85.75 ± 51.16 months) and had a collective total loss of just 3 teeth.

When comparing the progression of periodontal disease in these subgroups of patients all showed improvement of probeable pocket depth with associated attachment loss in all groups regardless of SPT frequency (Table 18). The greatest loss of attachment during maintenance was found in the “infrequent” group albeit the statistical power is small (only 8 patients). Nonetheless, this may be an indication that when patients are maintained infrequently there is a tendency for more attachment loss than when they are well maintained. Tooth loss was comparable for all groups except the “infrequent” group where the number of teeth lost was surprisingly lower (more favorable). This is even more remarkable when considering that this group also has the longest maintenance period (7 years).

We further investigated the different characteristics of these patients when dividing them into 2 major groups; compliant (q 3-4 months) and erratically/non-compliant (> 4 months) (Table 19). Although both groups in our study population showed similar improvement in attachment levels and probing depth measurements there were some significant differences. It appeared that when patients belonged in the severe disease
category they had a better chance of being compliant (p<0.01) The same applied for patients with deeper pockets (p<0.05) and with less favorable prognoses (p<0.01). It is also likely that all patients adhered to their suggested maintenance schedules but due to severity of the "compliant" group a more frequent SPT interval was suggested to them, hence they were seen more often in our study.

**TABLE No. 16**

<table>
<thead>
<tr>
<th>Characteristics of the Patient Population-2 (n =100)</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maintenance Effectiveness</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interval between SPT visits- Frequency (q months)</td>
<td>4.6 ± 1.12</td>
<td>3.29-9.62</td>
</tr>
<tr>
<td>Frequency of SPT visits</td>
<td>2.5/year</td>
<td></td>
</tr>
<tr>
<td>Number of months receiving Tx in Grad Perio</td>
<td>74.0 ± 40.6</td>
<td>30-233</td>
</tr>
<tr>
<td>Number of months in Initial Therapy</td>
<td>11.4 ± 5.1</td>
<td>3-30</td>
</tr>
<tr>
<td>Number of months in maintenance</td>
<td>62.6 ± 39.4</td>
<td>23-210</td>
</tr>
<tr>
<td>Months of maintenance for the Short-term pts (2-5 years)</td>
<td>36.8 ± 9.7</td>
<td>23-59</td>
</tr>
<tr>
<td>Months of maintenance for the Long-term pts (≥5 years)</td>
<td>103.1 ± 33.6</td>
<td>60-210</td>
</tr>
</tbody>
</table>

Table 16. Detailed presentation of the time intervals of each component of treatment delivered in our clinic

**TABLE No. 17**

**Distribution of Sample by Frequency of SPTs**

<table>
<thead>
<tr>
<th>Interval btw SPTs (months)</th>
<th>Number of patients</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3.0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Very Frequent 3.01-4.0</td>
<td>31</td>
<td>31</td>
</tr>
<tr>
<td>Frequent 4.01-5.0</td>
<td>45</td>
<td>45</td>
</tr>
<tr>
<td>Intermittent 5.01-6.0</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Infrequent &gt; 6.0</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>3.01-4.0 (3-4x/year)</td>
<td>31</td>
<td>31</td>
</tr>
<tr>
<td>4.01-6.0 (2-3x/year)</td>
<td>61</td>
<td>61</td>
</tr>
<tr>
<td>&gt; 6.0 (&lt; 2x/year)</td>
<td>8</td>
<td>8</td>
</tr>
</tbody>
</table>

Table 17. Patients were further divided into 4 groups based on the frequency with which they received professional cleanings.
### TABLE No. 18

**Disease Progression based on Frequency of SPTs**

<table>
<thead>
<tr>
<th>SPT Groups (q months)</th>
<th>Att. level during maint.</th>
<th>δ PD (improvement)</th>
<th>Tooth loss during maint.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very Frequent (n=31) 3.01-4.0</td>
<td>- 0.233</td>
<td>+0.166</td>
<td>1.10</td>
</tr>
<tr>
<td>Frequent (n=45) 4.01-5.0</td>
<td>- 0.066</td>
<td>+0.311</td>
<td>1.22</td>
</tr>
<tr>
<td>Intermittent (n=16) 5.01-6.0</td>
<td>- 0.013</td>
<td>+0.181</td>
<td>1.19</td>
</tr>
<tr>
<td>Infrequent (n=8) &gt; 6.0</td>
<td>- 0.381</td>
<td>+0.116</td>
<td>0.38</td>
</tr>
</tbody>
</table>

Table 18. After assigning our patients into groups based on SPT frequency we plotted the changes in clinical parameters (PD and AL) during the duration of maintenance for these groups. Tooth loss is also presented.

### TABLE No. 19

**Characteristics of Compliant Patients**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Compliance (SPT q 3-4 months)</th>
<th>Erratic Compliance or Non-Compliance (&gt; 4 months)</th>
<th>Difference (δ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of deep pockets (&gt; 7mm)</td>
<td>45%</td>
<td>33%</td>
<td>12%</td>
</tr>
<tr>
<td>Initial Prognosis</td>
<td>2.32</td>
<td>2.61</td>
<td>0.29</td>
</tr>
<tr>
<td>Severe periodontitis patients</td>
<td>77%</td>
<td>58%</td>
<td>19%</td>
</tr>
</tbody>
</table>

Table 19. This is a different distribution based on Wilson’s (1984) paper on compliance and erratic compliance. It suggests a pattern of compliant behavior based on the severity of the periodontal disease present.

### V.3.ii Periodontal stability of patients based on initial diagnosis

There are 64 patients with severe disease (64%) and 36 patients with moderate disease (36%). The patients with severe disease accounted for 83% of all teeth lost. They also lost nearly 3 times (OR: 2.75) more teeth than the patients with moderate disease. The extracted teeth from these two groups of patients differed significantly in initial PD (p<0.01), initial AL (p<0.01), initial prognosis (p<0.01) and mobility (p<0.01) (Table 20). The teeth extracted from the patients with moderate disease were more likely to
survive the initial phase and become extracted in maintenance (Table 21). They lost 15% of the extracted teeth (n=5) during active therapy and 85% in maintenance (n=29) (Appendix 3). The difference in number of extracted teeth between initial and maintenance phases was found significant (p<0.01).

The average tooth loss for the patients with severe disease was 40% higher during maintenance when compared to the patients with moderate disease (Figure 5). Tooth loss in the “severe” group was significantly higher in all three exam dates that were studied (initial, maintenance, final). What makes these differences even more important is the fact that the “severe” patients received SPT with similar frequency when compared to the “moderate” patients (q 4.48 vs. q 4.93 months) and actually were in maintenance for 1 year less than the “moderate” patients, i.e. 5 years vs. 6 years. They lost 50% of their extracted teeth during initial treatment (n=82) and 50% during maintenance (n=82) (Table 21). Those diagnosed with severe disease were 10 times more likely to lose a tooth during initial therapy.

The “severe” patients responded significantly more favorably in initial attachment gain (0.69 gain vs. 0.17 mm of loss) and decrease in probing depth (1.08 vs. 0.22 mm) (p<0.01) (Table 22). However, the PDs and attachment levels after initial Tx were still less favorable in the “severe” group. Both groups continued to show a decrease in their probing depths during maintenance. The “severe” patients had a decrease of 0.30 mm and the “moderate” had a decrease of 0.12 mm. Only the “severe” group continued to show mean attachment gain during maintenance (0.05 mm). The “Moderate” group continued
to present with attachment loss (0.31 mm). Nonetheless, both groups remained clinical stable during the maintenance phase.

The factors of smoking (31% vs. 25%), age (48y vs. 52y), sex (30% vs. 31% males), frequency of SPTs (q 4.5 months vs. q 4.9 months), were equally distributed among the two groups of “severe” and “moderate” patients. Only the duration of maintenance (4.8y vs. 5.9y) was different (See Appendices 4 & 5).

**TABLE No. 20**  
**Characteristics from Extracted Teeth of “Severe” and “Moderate” Patients**

<table>
<thead>
<tr>
<th>Characteristics (averages)</th>
<th>Extracted teeth from “severe” patients (n=64 patients)</th>
<th>Extracted teeth from “moderate” patients (n=36 patients)</th>
<th>Difference (δ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial PD</td>
<td>8.04 mm</td>
<td>5.62 mm</td>
<td>2.42 mm</td>
</tr>
<tr>
<td>Initial AL</td>
<td>8.76 mm</td>
<td>5.44 mm</td>
<td>3.31 mm</td>
</tr>
<tr>
<td>Initial Prognosis</td>
<td>1.88</td>
<td>2.44</td>
<td>0.56</td>
</tr>
<tr>
<td>Mobility at Init. Exam</td>
<td>1.33</td>
<td>0.82</td>
<td>0.51</td>
</tr>
</tbody>
</table>

Table 20. Analysis of the extracted teeth after they were divided into two groups based on the patients they were derived from, i.e. patients with either moderate or severe periodontal disease. Probing depths, attachment levels, mobility and initial prognosis are presented.

**TABLE No. 21**  
**Distribution of Tooth Loss in “Severe” and “Moderate” Patients**

<table>
<thead>
<tr>
<th>Characteristics (averages)</th>
<th>Tooth Loss in “Severe” patients (% within this group)</th>
<th>Tooth Loss in “Moderate” patients (% within this group)</th>
</tr>
</thead>
<tbody>
<tr>
<td>During Initial Tx</td>
<td>82 (50%)</td>
<td>5 (15%)</td>
</tr>
<tr>
<td>During Maintenance</td>
<td>82 (50%)</td>
<td>29 (85%)</td>
</tr>
<tr>
<td>During Entire Tx</td>
<td>164 (100%)</td>
<td>34 (100%)</td>
</tr>
</tbody>
</table>

Table 21. Analysis of the extracted teeth within each group, i.e. “severe” and “moderate” patients. The actual number of teeth is recorded within each group based on the time of the extractions (immediate or in maintenance). Next to each number a percentage is mentioned. Each percentage value given refers to that severity group only, i.e. within that column only.
Figure No. 5

Tooth Loss based on Initial Overall Diagnosis

![Graph analysis of the average tooth loss/patient in the 5.2 years of maintenance. Patients with moderate periodontal disease are plotted against those with severe disease.](image)

Table 22. Severe and moderate periodontitis patients are compared in the severity of their clinical parameters in the 3 different examinations of this study.
V.3.iii Changes of the periodontal condition in short term and long term patients

The Short Term (ST) patients (61) were maintained for 3.06 years while the Long Term (LT) patients (39) were maintained for 8.59 years (Table 23). After a year of initial therapy and 3 years of maintenance the ST patients showed a PD reduction of 0.84 and 0.19 mm respectively (overall PD reduction of 1.03mm). The LT patients (additional 5.6 more years of maintenance) showed an overall 0.97 mm reduction in PD. Both groups showed gains of attachment during initial therapy but during maintenance, the LT patients (8.6y) had an average loss of attachment of 0.32 mm vs. 0.03 mm in the ST patients. Notably, the LT patients lost twice as many teeth during maintenance but the duration of maintenance in this group was approximately 3 times longer (8.6y vs. 3y).

The factors of age (50y vs. 50y), sex (30% vs. 30% males), frequency of SPTs (q 4.8 months vs. q 4.5 months) were equally distributed between the two groups of LT & ST patients. However, there were differences within these groups regarding the distribution of smokers (36% LT vs. 25% ST) and severe patients (54% LT vs. 71% ST).

**TABLE No. 23**

Table 23. Comparison of long-term and short-term patients. Differences in probing depth, attachment level and tooth loss at initial exam, re-evaluation and final exam.

<table>
<thead>
<tr>
<th>Maintenance</th>
<th>Probing Depth</th>
<th>Attachment Level</th>
<th>Tooth Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>Re-Eval</td>
<td>Final</td>
</tr>
<tr>
<td><strong>Long Term</strong></td>
<td>4.94</td>
<td>4.27</td>
<td>3.97</td>
</tr>
<tr>
<td><strong>Short Term</strong></td>
<td>5.06</td>
<td>4.22</td>
<td>4.03</td>
</tr>
</tbody>
</table>
V.3.iv Effect of smoking on clinical outcome

There were 34 current and previous smokers and 66 nonsmokers. Smokers and nonsmokers were similar in age, sex distribution, frequency of SPTs, disease severity and duration of maintenance (Table 24).

Smokers (S) showed an overall average improvement in pocket depths during the initial phase of treatment (0.92 mm), which continued to increase during maintenance by 0.03 mm (overall improvement of 0.95 mm) (Table 25). Nonsmokers (NS) showed 0.71 mm reduction in PD, which continued during maintenance (0.29 mm) and lead to an overall PD reduction of 1.0 mm. Both groups showed similar PD improvement. Although smokers showed a more favorable attachment gain during initial treatment they also exhibited a greater attachment loss during maintenance (Appendix 2). Overall, smokers had an average attachment gain of 0.18 mm vs. 0.25 mm for the nonsmokers. Smokers were more likely to loose teeth during both initial therapy and maintenance phases. However, the differences are not significant which indicate that both smokers and nonsmokers were well maintained at the UBC graduate clinic.

Although there were no significant differences between smokers and nonsmokers when comparing moderate pockets, this was not the case with deep pockets. In the deeper pockets, nonsmokers showed significantly better PD reduction (0.83 mm) during maintenance compared to smokers (0.36 mm) (p<0.05) (Figure 6). Attachment loss was also significantly different. Although showing similar gains during the initial phase of
treatment, the overall gain in attachment in deep pockets (≥7 mm) for nonsmokers was 1.47 mm vs. 0.77 in smokers (p<0.05).

TABLE No. 24
Population Characteristics between Smokers and Nonsmokers

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex % males/females</th>
<th>Frequency of SPTs</th>
<th>Severity of disease</th>
<th>Duration of maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smokers</td>
<td>45.4</td>
<td>34.5/ 65.5</td>
<td>q 4.5 months</td>
<td>69% severe</td>
</tr>
<tr>
<td>Nonsmokers</td>
<td>51.8</td>
<td>31.8/ 68.2</td>
<td>q 4.7 months</td>
<td>59% severe</td>
</tr>
</tbody>
</table>

Table 24. Smokers and nonsmokers are compared in certain characteristics to confirm uniformity of these groups. Age, gender, SPT frequency, severity of periodontal disease and duration of maintenance are mentioned.

TABLE No. 25
Disease Progression based Smoking Status

<table>
<thead>
<tr>
<th>Maintenance</th>
<th>Probing Depth</th>
<th>Attachment Level</th>
<th>Tooth Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial</td>
<td>Re-Eval</td>
<td>Final</td>
</tr>
<tr>
<td>Smokers</td>
<td>5.32</td>
<td>4.40</td>
<td>4.37</td>
</tr>
<tr>
<td>Nonsmokers</td>
<td>4.88</td>
<td>4.17</td>
<td>3.88</td>
</tr>
</tbody>
</table>

Table 25. Comparison of smoking and nonsmoking patients. Differences in probing depth, attachment level and tooth loss at initial exam, re-evaluation and final exam are plotted.
Figure 6
Change in Clinical Parameters of Surviving Teeth with Deep PDs; Smokers Vs. Nonsmokers

Figure 6. Comparison of probing depth changes in smokers and non smokers in pockets with initially deeper measurements, i.e. ≥7 mm. Dark lines represent the smokers while the light lines represent the non smokers. Continuous lines represent probing depths while interrupted lines represent the attachment levels of those deeper pockets.
V.4 Prognosis vs. Tooth Survival

In our graduate clinic study 63% of the total patient population lost no teeth during the first year of treatment (initial phase) while 35% of patients lost no teeth during initial therapy and maintenance (Table 26). Notably, 28% of them lost no more than 2 teeth during the study period while 4 patients (4%) lost 9-10 teeth. Two of the latter patients lost all 9-10 teeth during the initial therapy phase.

Tooth loss was examined in relation to initial prognosis and nearly 25% of all teeth that were lost were given a hopeless prognosis (Figure 7) (Appendix 5). This is significant because only 2.3% of all teeth were given this prognosis. However, the majority of teeth that were lost, i.e. 56.8% were originally given a poor prognosis. The chance of having a tooth extracted was associated with its initial prognosis, and length of survival during maintenance. 75% of all hopeless teeth that were eventually lost, were lost during the initial phase of treatment while the remaining 25% were lost during maintenance (Table 27). Thirty six percent (36%) of all extracted teeth with a poor prognosis were lost during active therapy. This means that 64% of the poor teeth that were eventually extracted survived the initial therapy phase. Only 20% of the fair teeth that were lost were extracted during the initial treatment period. 80% of the extractions of fair teeth were performed during maintenance.
## Table No. 26

### Study Population Classified by Tooth Loss

<table>
<thead>
<tr>
<th>Tooth loss while in treatment</th>
<th>Number of patients</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial Tx</td>
<td>Maintenance</td>
<td>Final</td>
<td></td>
</tr>
<tr>
<td>0 teeth</td>
<td>63 (63%)</td>
<td>56 (56%)</td>
<td>35 (35%)</td>
<td></td>
</tr>
<tr>
<td>1-2 teeth</td>
<td>28 (28%)</td>
<td>29 (29%)</td>
<td>37 (37%)</td>
<td></td>
</tr>
<tr>
<td>3-4 teeth</td>
<td>5 (5%)</td>
<td>9 (9%)</td>
<td>15 (15%)</td>
<td></td>
</tr>
<tr>
<td>5-6 teeth</td>
<td>1 (1%)</td>
<td>3 (3%)</td>
<td>7 (7%)</td>
<td></td>
</tr>
<tr>
<td>7-8 teeth</td>
<td>1 (1%)</td>
<td>3 (3%)</td>
<td>2 (2%)</td>
<td></td>
</tr>
<tr>
<td>9-10 teeth</td>
<td>2 (2%)</td>
<td>0</td>
<td>4 (4%)</td>
<td></td>
</tr>
<tr>
<td>&gt; 10 teeth</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

86 teeth lost 112 teeth lost 198 teeth lost

Table 26. Distribution of patients according to the number of teeth they lost during initial treatment, maintenance and overall. Each column represents percentages within that specific examination. Therefore, each column sums up to a total of 100%.

### Figure No. 7

#### Distribution of Total Tooth Loss based on Initial Prognosis

Figure 7. Bar graph distributing all extracted teeth into 4 categories according to their initial prognosis, i.e. good, fair, poor, hopeless.
TABLE No. 27
Distribution of Tooth Loss (Initial vs. Maintenance Phase) based on Prognosis

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Number of teeth Lost</th>
<th>Lost initially</th>
<th>Lost in Maintenance</th>
<th>% lost during Initial Tx</th>
<th>% lost during Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopeless</td>
<td>49</td>
<td>37</td>
<td>12</td>
<td>75.51</td>
<td>24.49</td>
</tr>
<tr>
<td>Poor</td>
<td>113</td>
<td>41</td>
<td>72</td>
<td>36.28</td>
<td>63.72</td>
</tr>
<tr>
<td>Fair</td>
<td>30</td>
<td>6</td>
<td>24</td>
<td>20.0</td>
<td>80.0</td>
</tr>
<tr>
<td>Good</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>42.86</td>
<td>57.14</td>
</tr>
</tbody>
</table>

Table 27. The extracted teeth only are grouped according to their pretreatment prognosis. Tooth loss is compared in the different examination time points. The last two columns represent the time distribution of tooth loss within each category as a percentage. Each row totals 100% in these last 2 columns.

All teeth were studied based on their pretreatment prognosis and survival at an average of 5.22 years of maintenance. When a tooth was given a hopeless prognosis it had a 64% chance of being extracted during initial therapy (during the first year). This number increased to 85% of a likelihood of extraction when the 5-year maintenance period was examined. Interestingly, nearly 80% of those teeth initially classified as poor, survived at 5 years (Table 28). We divided our population into two groups, i.e. those who were maintained for less than 8 years (n = 81) and those that were in maintenance for 8 years or longer (n = 19) (Appendix 4). This subgroup (19 subjects) of longer-maintained patients had 21% smokers (vs. 29% of the general population) and 73% nonsmokers (vs. 66% of the total study sample). They received 12.3 months of initial therapy (vs. 11.3 of all patients) and had an average of 11 years of maintenance (10.83 years). Gender was also similarly distributed compared to the entire study population.

In this group of longer-maintained patients (≥8 years) the likelihood of a hopeless tooth being extracted increased to 90%, while a poor tooth had a 32% chance of being
extracted within 11y of maintenance (Table 29). However, with treatment and maintenance in the graduate periodontics clinic, teeth that are originally given a poor prognosis have an 81% chance of surviving a 4-year maintenance period (n= 81) and a nearly 70% chance of surviving with 11 years of maintenance (n= 19). Generally when a tooth is given a less optimistic prognosis the likelihood of it being extracted increased at each time-point examined (Figures 8,9,10).

TABLE No. 28
Pretreatment Prognosis vs. Tooth Loss (All Patients)

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Number of teeth</th>
<th>Lost initially</th>
<th>Lost in Maintenance</th>
<th>Total Lost</th>
<th>% lost from each prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopeless</td>
<td>58</td>
<td>37 (63.79%)</td>
<td>12</td>
<td>49</td>
<td>84.48</td>
</tr>
<tr>
<td>Poor</td>
<td>541</td>
<td>41 (7.58%)</td>
<td>72</td>
<td>113</td>
<td>20.89</td>
</tr>
<tr>
<td>Fair</td>
<td>685</td>
<td>6 (0.88%)</td>
<td>24</td>
<td>30</td>
<td>4.38</td>
</tr>
<tr>
<td>Good</td>
<td>1228</td>
<td>3 (0.24%)</td>
<td>4</td>
<td>7</td>
<td>0.57</td>
</tr>
</tbody>
</table>

Table 28. Number of teeth and their losses according to their initial prognosis. The two columns with percentages represent the percent of teeth lost from the entire tooth population of that prognosis category.

TABLE No. 29
Pretreatment Prognosis vs. Tooth Loss (Longer-Maintained pts, ≥8 years)

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Number of teeth</th>
<th>Lost initially</th>
<th>Lost in Maintenance</th>
<th>Total Lost</th>
<th>% lost from each prognosis when ≥ 8y of maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopeless</td>
<td>10</td>
<td>7 (70.00%)</td>
<td>2</td>
<td>9</td>
<td>90.00</td>
</tr>
<tr>
<td>Poor</td>
<td>91</td>
<td>11 (12.09%)</td>
<td>18</td>
<td>29</td>
<td>31.87</td>
</tr>
<tr>
<td>Fair</td>
<td>122</td>
<td>1 (0.82%)</td>
<td>12</td>
<td>13</td>
<td>10.66</td>
</tr>
<tr>
<td>Good</td>
<td>229</td>
<td>2 (0.87%)</td>
<td>0</td>
<td>2</td>
<td>0.87</td>
</tr>
</tbody>
</table>

Table 29. This table contains data only from a subgroup of our patients (19 patients) that had an average of 11 years in maintenance. The number of teeth and their losses according to their initial prognosis are presented. The two columns with percentages represent the percent of teeth lost from the entire tooth population of that prognosis category.
Figure No. 8

Tooth Survivability based on Initial Prognosis

![Bar graph showing survival rates](image)

Figure 8. This bar graph represents the percentage of teeth that survived in maintenance. Teeth are pooled into prognosis categories. The data for the initial visit (time= -1) and just after completion of active therapy (t= 0) were taken from the entire study sample (100 patients). Then we divided patients into those who received treatment < 8 years (average of 4 years, n=81) and those who received treatment ≥ 8 years (average of 11 years, n=19).
Figure No. 9
Tooth Survivability based on Initial Prognosis (Average of 5.2y in 100 pts)

Figure 9. This graph represents the percentage of teeth that survived in maintenance. All teeth have been included into the 4 different prognosis categories. Data from all 100 patients were used.

Figure No. 10
Tooth Survivability based on Initial Prognosis (Average of 11y in 19 pts)

Figure 10. This graph represents the percentage of teeth that survived in maintenance. All teeth have been pooled into prognosis categories. The data values used were taken from a longer-maintained (11 years) subpopulation of 19 patients.
Since molars are more commonly lost than nonmolar teeth, we examined them further in relation to their survivability and prognosis. Our data lead to the suggestion that the “worse” the initial prognosis is for a molar the more likely it will have deeper probing depths and increased attachment loss. Molars with different prognoses exhibited significantly different initial probing depth and attachment loss measurements (p<0.05) (Table 30). Molars that were assigned a good or fair prognosis showed 98.6% and 96.8% survivability at 5 years. Interestingly, nearly 80% (77.1%) of poor molars survived at 5 years (Figure 11).

**TABLE No. 30**

**Initial Prognosis and Disease Progression (Molars only)**

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Initial PD</th>
<th>Initial AL</th>
<th>Survivability at 5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopeless</td>
<td>8.5</td>
<td>10.0</td>
<td>13.8 %</td>
</tr>
<tr>
<td>Poor</td>
<td>6.8</td>
<td>7.2</td>
<td>77.1 %</td>
</tr>
<tr>
<td>Fair</td>
<td>5.6</td>
<td>5.6</td>
<td>96.8 %</td>
</tr>
<tr>
<td>Good</td>
<td>4.6</td>
<td>3.6</td>
<td>98.6 %</td>
</tr>
</tbody>
</table>

Table 30. Clinical characteristics of the different prognosis categories and the survivability they presented. This table refers to molar teeth only.
Figure 11. This graph represents the percentage of molars that survived in maintenance. Molars are grouped into prognosis categories. The data values used were taken from the entire study sample (100 patients).
V.5 Molar Survivability

V.5.i Molar Survivability and Furcation Involvement

Notably, 90.1% of all molars survived for the average 5-year span of our maintenance program. When investigating the furcated molars alone the survivability was still high, i.e. 80.5% (389 out of 483 furcated molars) (Table 31). There weren’t enough molars to graph an 11-year analysis.

The survivability of these furcated molars differed depending on degree of furcation (Figure 12). For example the percentage of furcated molars that were still present at the end of our study period was 94.5% for class-I furcated molars and dropped to 53.3% for class-III furcated molars. It becomes apparent that the degree of furcation should play an important role when assigning a prognosis in a molar.

**TABLE No. 31**

<table>
<thead>
<tr>
<th>Furcation Classification</th>
<th>Extracted Molars (n &amp; %)</th>
<th>Surviving Molars (n &amp; %)</th>
<th>All Molars (n &amp; %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>20 (14.9%)</td>
<td>114 (85.1%)</td>
<td>134 (100%)</td>
</tr>
<tr>
<td>Class-I</td>
<td>10 (5.5%)</td>
<td>171 (94.5%)</td>
<td>181 (100%)</td>
</tr>
<tr>
<td>Class-II</td>
<td>56 (23.1%)</td>
<td>186 (76.9%)</td>
<td>242 (100%)</td>
</tr>
<tr>
<td>Class-III</td>
<td>28 (46.7%)</td>
<td>32 (53.3%)</td>
<td>60 (100%)</td>
</tr>
<tr>
<td>SUM</td>
<td>114</td>
<td>503</td>
<td>617</td>
</tr>
</tbody>
</table>

Table 31. Molars are pooled into categories according to the degree of furcation they presented at the time of their initial exam. The 1st column shows the different furcation classes. Column 2 presents the number of total extractions within each furcation class. Column 3 presents the number of molars that survived the duration of our study while the last column presents the total sum of molars that were presented at the initial exam within each degree of furcation.
Figure No. 12

Molar Survivability based on Furcation Involvement

Figure 12. This graph represents the percentage of molars (only) that survived initial therapy and maintenance. The molars are pooled into the 3 different furcation categories. The values plotted on this graph are taken from the entire study sample (100 patients).
V.5.i Molar survivability and mobility involvement

Mobility likely influences the decision for extraction of molars as well and possibly more than furcation involvement. Over 90% of non-mobile molars survived our 5-year study (Table 32). However, the survival of the mobile molars decreases drastically as the degree of mobility increases. Although molars with class-I mobility present 77% survivability at 5 years, this number declines to 50% and 15% for class-II and class-III molars respectively (Figure 13). Notably within the phase of active therapy 85% of molars with a mobility grading of 3, are extracted. Again, the number of molars that survived 11 years was not adequate to analyze further, hence there is not an 11-year analysis graph in this section.

TABLE No. 32
Survivability of Molars in Relation to their Degree of Mobility

<table>
<thead>
<tr>
<th>Mobility Classification</th>
<th>Extracted Molars (n &amp; %)</th>
<th>Surviving Molars (n &amp; %)</th>
<th>All Molars (n &amp; %)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>40 (9.5%)</td>
<td>380 (90.5%)</td>
<td>420 (100%)</td>
</tr>
<tr>
<td>Class-I</td>
<td>27 (23.1%)</td>
<td>90 (76.9%)</td>
<td>117 (100%)</td>
</tr>
<tr>
<td>Class-II</td>
<td>30 (50%)</td>
<td>30 (50%)</td>
<td>60 (100%)</td>
</tr>
<tr>
<td>Class-III</td>
<td>17 (85%)</td>
<td>3 (15%)</td>
<td>20 (100%)</td>
</tr>
<tr>
<td>SUM</td>
<td>114</td>
<td>503</td>
<td>617</td>
</tr>
</tbody>
</table>

Table 31. Molars are pooled into categories according to the degree of mobility they presented at the time of their initial exam. The 1st column shows the different mobility classes. Column 2 presents the number of total extractions within each mobility class. Column 3 presents the number of molars that survived the duration of our study while the last column presents the total sum of molars that were presented at the initial exam within each degree of mobility.
Figure No. 13

Molar Survivability based on Degree of Mobility

Figure 13. This graph represents the percentage of molars (only) that survived initial therapy and maintenance. The molars are pooled into the 3 different mobility categories. The values plotted on this graph are taken from the entire study sample (100 patients).
Chapter Six  Discussion

VI.1 Materials and Methods

This is a retrospective study of 100 subjects selected consecutively from the maintenance patient pool (1997 to 2001). All patients were in maintenance for a minimum of 2 years. In this study design all prognoses were determined by a single examiner, i.e. Dr. Irinakis, based on radiographs, probing depths and attachment levels. This differs from some long term studies in that a prognosis is given without the clinical picture of the patient studies (Ramfjord et al., 1973; Ramfjord et al., 1980; Pihlstrom et al., 1983; Nabers et al, 1988). However, it compares favorably to other retrospective long term maintenance studies. For example Becker et al (1984) used periapical radiographs and probing depths to assign the prognoses while Checchi et al (2002) used periapical radiographs alone. We included the use of attachment levels in our decision making, which increases the validity of our prognoses. It is necessary to realize the true extent of the disease on any given tooth. Probing depth measurements alone may be somewhat deceiving since inflammation induced pseudopocketing is commonly present at the initial exam and they don’t provide information on pre-existing recession. This is crucial when assigning a pre-treatment prognosis. The additional criteria for assigning an individual tooth prognosis were based on the studies of McGuire (1991) and McGuire & Nunn (1996).
VI.2 Clinical Parameters and Maintenance

PROBING DEPTHS AND ATTACHMENT LEVELS

Our average maintenance period extends to 5.22 years, which satisfies the criteria of Ramfjord (1973) in that to truly investigate long term differences in periodontal clinical parameters, maintenance studies should exceed 3 years.

Significant improvements of mean clinical measurements took place as a result of initial therapy and prescribed maintenance. Our initial improvement (during active therapy) of probing depths and attachment levels in the deeper pockets (improvement of 2.2 mm and 1.5 mm, respectively) compare favorably to other studies that showed initial improvement of 1.51-2.22 mm (PD) and 0.91-1.4 mm (AL) respectively (Morrison et al., 1980; Pihlstrom et al., 1981; Kaldahl et al., 1988). In agreement with these studies our improvement in the deeper pockets was considerably more favorable than the observed improvement in the 4-6 mm pockets.

During maintenance we noticed a slight deterioration in attachment levels in the moderate and deeper pockets at 5 years. Other studies of similar maintenance duration (4-5 years) presented an initially significant improvement in attachment levels that also slightly deteriorated in maintenance (Pihlstrom et al., 1981; Knowles et al., 1979; Renvert et al., 1990). Collectively, these data indicate the effectiveness of our maintenance program.

The impact that tooth type had on disease stability produced similar results with a 6.5-year study by Pihlstrom et al (1984). For example, we observed a profound positive
effect initial and SPT treatment had on the deeper nonmolars. Probing depth improvement of 3.22 mm and attachment gains of 2.02 mm were sustained over the maintenance period of 5.22 years. These values agree with Pihlstrom who had a 4 mm PD decrease and a 1.53 mm attachment gain. However, in the deeper molars he showed similar PD improvement (over 2 mm) and he had a significantly better response in attachment levels. Similarities and differences between these two studies are observed in the moderate pockets as well. Perhaps the reason behind this discrepancy with the molars lies in the number of subjects. Our study evaluated data collected from 100 patients and 2,500 teeth while Pihlstrom’s study evaluated 10 patients and just over 260 teeth. The data from our study suggests that even severely involved multirooted teeth showed stability of the treatment outcome when properly maintained.

TOOTH LOSS

In a 14-year long term maintenance study it was shown that more tooth loss was recorded as the duration of the study increased (Lindhe and Nyman, 1984). The same pattern occurred in our study. When tooth loss increased from 0.75 (per patient) at 3 years to 1.12 at 5 years to 1.67 and 1.68 (at 8.5 and 11 years respectively). This is not far off from other studies that showed an average tooth loss/patient 1.0 in 6 years of a well-maintained population (Kocher et al., 2000) to 1.6 in 6.5 years (Becker et al., 1984) to 2.6 in 19 years (McFall, 1982). However, there are studies that have shown remarkable results in their maintenance such as 1.8 teeth lost/patient in 22 years (Hirschfeld and Wasserman (1978) or 0.49 in 14 years (Lindhe and Nyman, 1984) or even 0.29 in 12.9 years (Nabers et al., 1988). In the first study the difference is the severity of the study sample. Hirschfeld and
Wasserman had a number of cases with mild periodontal diseases. Our study included only moderate to advanced periodontally involved patients (mostly advanced). In the second study the main difference is the average number of teeth to begin with. In our study there was an average of over 24 teeth at maintenance while Lindhe and Nyman after a vigorous active therapy phase they maintained a population with an average of 21.8 teeth. This might just be an indication of either a more aggressive approach during active treatment in their study or a more conservative approach in our study. In Nabers private practice study there is not enough information given on his article in order to make comparisons. From the above it becomes evident that our study is comparable to other long term studies in North America and Europe. We can assume that the tooth loss displayed by our population sample falls within the norm and that our maintenance program has been able to control the necessity for extraction.

Approximately 1/3 of the patients (34%) lost no teeth at all while 15% of tooth loss was concentrated on 3 patients (3% of patients). This concentration of extreme tooth loss on a few patients is often found in the literature (Hirschfeld & Wasserman, 1978; McFall, 1982; Nabers et al., 1988). Since the reasons for extractions in our study could not be determined in retrospect, it is possible that other factors (other than periodontal) played a role. Regardless, the tooth loss ranking in our study is very similar to other classic longitudinal retrospective studies (Hirschfeld & Wasserman, 1978; McFall, 1982). In both of these studies and in our study the teeth most likely to be lost are the maxillary and mandibular molars followed by the maxillary bicuspids and the mandibular central. Our main difference with these studies is the tooth that outsurvived all others, which in our
study was the maxillary central in contrast to the mandibular canine in these other studies. Basically our investigation has shown important similarities with these well-known tooth loss studies.

Our tooth extraction data show that during maintenance the majority of extracted teeth were molars (60%). This percentage is identical to that found in the long-term maintenance study of Hirschfeld and Wasserman (1978). When analyzing the data further it became obvious that certain factors seem to strongly influence the likelihood and the timeline of these molar extractions. Furcation and mobility seem to play an important role in determining whether a molar will become extracted. If it does, then the severity of furcation and/or mobility involvement will also determine the time of the extraction, i.e. either immediate or during maintenance. Although 11.9% of all molars were extracted, this number increased to 19.5% of all furcated molars being lost in a 5-year span. This number is significantly lower when compared to other long term studies. Pearlman lost 26.5% of furcated teeth. Hirschfeld and Wasserman lost 29.5% while McFall lost 58% of furcated molars. Although we might be tempted to conclude that our maintenance schedule showed better results than these other studies it is important to acknowledge that our study was a 5 year study when their studies were 10, 22 and 19 years of duration, respectively. It has already been shown that longer durations of maintenance is accompanied by higher losses of teeth. Analyzing our data further in our study we found that the more involved the furcation was the higher number of extractions was performed. Only 5.5% of molars with class-I furcations were extracted compared to 23.1% and 46.7% as the involvement became class-II and class-III. This confirms the assumption
that assignment of a prognosis should take into account furcation involvement as well. Mobility also played a significant role in our decision making for extraction. Our data indicate that the likelihood of a molar being extracted increases with increasing mobility grading. Apparently 65% of all molars with class-II mobility or greater were extracted. Only 12.5% of molars with class-I mobility or less were extracted. It seems logical to conclude that based on our findings tooth mobility and degree of furcation are factors that significantly influence our decision for tooth extraction. This concept is in agreement with Svardstrom and Wennstrom (2000) who conducted a study on factors influencing treatment decision for molars.

DURATION OF MAINTENANCE/ FREQUENCY OF SPT/COMPLIANCE

Our sample population of 100 patients was seen for an average duration of maintenance of 5.2 years. This is considered to be a long-term maintenance population. They were seen on average every 4.5 months. The greatest majority of our patients (92%) were seen every 3-6 months. This frequency of SPT visits suggests that our graduate clinic is maintaining our patients well. The 3-6 month interval between professional debridement visits is considered by many clinicians optimal for an average periodontally involved population (McFall, 1982; Nabers et al., 1988; Pearlman, 1993; Kocher et al., 2000). All of our patients showed similar stability of attachment levels and probing depths so long as they were within that 3-6 months interval. Even tooth loss was comparable. It is accepted that not all periodontal patients need to be seen (after completion of surgical phase of treatment) 4 times a year. However, the greatest loss of attachment was observed in our infrequently maintained subgroup (8 patients seen later than every 6 months)
although the small sample size does not allow us to draw any definitive conclusions. Other studies however, have clearly shown that patients who adhere to a suggested maintenance intervals loose fewer teeth and exhibit greater improvement in pocket depths and attachment gain (Wilson et al., 1987; Checchi et al., 2002). When attempting to follow Wilson’s criteria for compliance (1996) we divided our population into groups with 3-4 month SPT intervals and with over 4 month SPT intervals. We observed that the “more compliant” group had 50% more “deeper” pockets (≥7mm) and 33% more severe patients than the “less compliant” group. They also had a poorer prognosis. One could interpret this as that when patients were more severe and were given a poorer prognosis they had a better chance of being compliant. However, after evaluating the data further it became apparent that both groups showed similar improvement in attachment levels and probing depth measurements. This lead us to believe that perhaps both groups were “compliant” to their suggested maintenance schedules and perhaps these schedules just differed due to the difference in initial disease severity. In conclusion, if patients adhere to their maintenance schedules then the results of active therapy can be maintained in the long term. Ramfjord (1982) came to the same conclusion and actually showed that with proper maintenance initial therapy results can be maintained even in the presence of less than optimal oral hygiene.
VI.3 Smoking

Our groups of smokers and nonsmokers were well balanced at the time of initial examination in regard to age, gender, SPT frequency, disease severity and duration of maintenance. In the present study, smokers (29% of population) had a higher percentage of deeper pockets. This is in agreement with Linden and Mullally (1994) who also showed that the number of deeper pockets and deeper attachment levels were significantly associated with smoking.

The present study shows similar initial improvement in probing depth and attachment levels. It seems that the response to the initial phase of treatment, which included SRP and surgery, was similar for smokers and nonsmokers in our clinic. Kinane and Radvar (1997) also found a similar initial response in attachment gain but not in probing depth resolution. Their study showed better results for the nonsmokers even at the initial phase. When comparing the readings during maintenance in our study it became apparent that differences did exist between smokers and nonsmokers, which were significant in the deeper pockets. In these more involved sites (≥ 7mm PD), nonsmokers exhibited a significantly superior response in both probing depths and attachment levels. This is in agreement with other studies that showed that pocket depth resolution was greater in nonsmokers and this difference increased as the pocket depth increased (Preber and Bergstrom, 1990; Grossi et al., 1996). This lead us to the suggestion that it may be beneficial to our maintenance program if we included a strict smoking cessation protocol that would be part of every recall exam and not limit our focus to smoking only during
the initial treatment. Constant re-enforcement may level these differences found in the
deeper sites. Regardless of these differences in the deeper sites, there were no significant
differences in tooth loss. It seems that although there are differences in the amount of
improvement in PD and AL, our program is still able to contain tooth loss (regardless if
they belong to smokers or not). Perhaps if our study had a longer duration then
differences may have emerged between these two groups in matters of tooth loss. Other
studies seem to support this idea by showing significant differences in tooth loss between
smokers and nonsmokers (Holm, 1994; Albandar et al., 2000; Jansson and Lavstedt,
2002).

VI.4 Tooth Survival and Prognosis

OVERALL TOOTH SURVIVABILITY

Hirschfeld and Wasserman (1978) in their classical study of tooth loss over a period of 22
years had a 7.1% tooth loss during their maintenance period. McFall (1982), using the
same criteria showed 9.9% of tooth loss during maintenance. They extracted 60% and
52%, respectively of all questionable teeth during maintenance. They had divided all
teeth into just two prognosis categories, i.e. questionable and favorable. The criteria are
limited and not clearly stated. Their results make it difficult to distinguish any true
differences between questionable and favorable teeth in matters of percentage of loss.
Perhaps a different prognosis assignment approach would be necessary. At UBC we
follow the criteria set by McGuire and Nunn (1991) with slight modifications (see
Materials and Methods section) so that in the end we only use 4 prognosis categories.
instead of the 5 they had suggested. In the present study we found the following survival rates for our prognosis categories at 5 years; 99.4% for good, 95.6% for fair, 79.1% for poor and 15.5% for hopeless. These numbers seem to compare rather well with McGuire and Nunn (1996) in their study of 100 patients where they used 5 prognosis categories, i.e. good, fair, poor, questionable, and hopeless. They found 97.9%, 92.1%, 86.6%, 45% and 38% survival rates respectively (Table 33). However, their study had an average of 10 years in maintenance. It would seem that our study has very close survival rates for all prognosis categories other than hopeless teeth. In order to get a better sense of comparison we used the data from our subpopulation that were in maintenance for 11 years. The survival rates changed to 99.1%, 89.3%, 68.1% and 10% (Table 34). Our similarities remained for mostly the good and fair prognosis while the poor and hopeless drew further apart. Since we used similar criteria for prognosis assignment we investigated these differences further. A rather important difference seems to be in the poor prognosis category. It would appear initially that our prognosis assignment (with its modification to the criteria set by McGuire and Nunn) leads us to a more strict approach when assigning a poor prognosis (meaning that when it is borderline between fair and poor we would assign a poor prognosis), hence this noticeable difference in survivability between our poor and fair prognosis group. McGuire and Nunn only have a 5.5% difference in survivability between fair and poor after 10 years in maintenance. Our modification lead to assigning a poor prognosis to all teeth that would be prognosticated as questionable. This seems to have served us well and lead to a more “appropriate” assignment of a poor prognosis where there is an “actual” difference in survivability between poor and fair. When looking into their data again and pooling the poor and
questionable teeth together (as per our modified criteria) the survival rate of the poor teeth drops to 79% at 10 years (Table 35). The actual difference between poor and fair now becomes 13%, which is closer to our 16% difference at 5 years (21% difference at 11 years). It is difficult to compare the hopeless teeth with their study since the extractions performed in this category is highly patient driven in a sense that the numbers are highly dependent on how often the patients follow our recommendation for extraction of these teeth. There is a lot of controversy when it comes to extracting teeth. It has been shown that when hopeless teeth are retained and no periodontal treatment is delivered then the adjacent teeth will loose attachment with a rate 10 times that of the adjacent teeth that had those hopeless teeth extracted (Machtei et al., 1989). However, in a well maintained population it has been shown that retained hopeless teeth do not have any significant detrimental effect to their adjacent teeth for up to 8 years (DeVore et al., 1988; Wojcik et al., 1992). It is reasonable to assume that perhaps an effort could be made in certain occasions to maintain hopeless teeth in periodontitis patients.

In the end, the results from our study indicate that the criteria set from our graduate periodontics clinic in assigning a prognosis are valid and that it seems reasonable to limit ourselves to only 4 prognosis categories rather than 5.
TABLE No. 33
Comparison of Survivability between McGuire & Nunn and UBC-1.

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Survivability at 10 years (McGuire &amp; Nunn, 1996)</th>
<th>Survivability at 5 years (UBC study, 2003)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopeless</td>
<td>38 %</td>
<td>15.5 %</td>
</tr>
<tr>
<td>Questionable</td>
<td>45 %</td>
<td>N/A</td>
</tr>
<tr>
<td>Poor</td>
<td>86.6 %</td>
<td>79.1 %</td>
</tr>
<tr>
<td>Fair</td>
<td>92.1 %</td>
<td>95.6 %</td>
</tr>
<tr>
<td>Good</td>
<td>97.9 %</td>
<td>99.4 %</td>
</tr>
</tbody>
</table>

Table 33. Comparison of the survivability between these two studies of 100 patients each. Data from all patients present.

TABLE No. 34
Comparison of Survivability between McGuire & Nunn and UBC-2.

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Survivability at 10 years (McGuire &amp; Nunn, 1996)</th>
<th>Survivability at 11 years (UBC study, 2003)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopeless</td>
<td>38 %</td>
<td>10%</td>
</tr>
<tr>
<td>Questionable</td>
<td>45 %</td>
<td>N/A</td>
</tr>
<tr>
<td>Poor</td>
<td>86.6 %</td>
<td>68.1 %</td>
</tr>
<tr>
<td>Fair</td>
<td>92.1 %</td>
<td>89.3 %</td>
</tr>
<tr>
<td>Good</td>
<td>97.9 %</td>
<td>99.1 %</td>
</tr>
</tbody>
</table>

Table 34. Comparison of the survivability between these two studies. Our data are taken from a subpopulation of 19 patients that were maintained for an average of 11 years.

TABLE No. 35
Comparison of Survivability between McGuire & Nunn and UBC-3.

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Survivability at 10 years (McGuire &amp; Nunn, 1996)</th>
<th>Survivability at 5 years (UBC study, 2003)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopeless</td>
<td>38%</td>
<td>15.5 %</td>
</tr>
<tr>
<td>Poor</td>
<td>79%</td>
<td>79.1 %</td>
</tr>
<tr>
<td>Fair</td>
<td>92.1%</td>
<td>95.6 %</td>
</tr>
<tr>
<td>Good</td>
<td>97.9%</td>
<td>99.4 %</td>
</tr>
</tbody>
</table>

Table 35. The data from the "poor" and "questionable" teeth have been pooled together in McGuire & Nunn's study for purposes of comparison with our study. Data from all patients in both studies presented.
MOLAR SURVIVABILITY

In an effort to accumulate more data on the relationship that certain factors had on the survivability of molars in our study we investigated the impact of furcation and mobility and compared our results to McGuire and Nunn (1996) that performed a very similar investigation with similar number of patients and teeth. We found survivability rates of 94.5%, 76.9% and 53.3% in class-I, II and III furcated molars respectively in 5.2 years of average maintenance. These numbers compare favorably to those by McGuire, i.e. 92.1%, 82.4% and 70.3%. They seem to have maintained their class-III furcated molars significantly better than we did in our study. However, it would be difficult to come to any definitive conclusions because we would have to know what percentage of those molars were also associated with a higher degree of mobility. It seems that furcations affect the survivability of molars to a significant extent. The mode of treatment has to be carefully planned when treating furcated molars in order to increase their survivability. It has been shown that maxillary 2nd premolars adjacent to a class-II furcation involvement, when treated with only scaling and root planing show a significantly less favorable probing depth reduction (by 0.5 mm) than their mesial site (Ehnevid H, Jansson LE, 2001). There seems to be an inhibitory effect of maxillary furcations to their adjacent second premolars and perhaps a surgical approach may be considered when addressing such situations.

When comparing the impact of mobility (on its own) we had a molar survivability of 76.9%, 50% and 15% in class-I, II and III mobile molars. McGuire and Nunn showed a survivability of 83%, 73% and 53%. Again there are subtle differences that may explain
in part this outcome. Although both studies (ours and theirs) had similar number of patients and teeth, they had a less severely involved periodontal population than our study, i.e. 71% of good teeth vs. 48.8% of good teeth in our study.
Conclusions

1. The maintenance program of the graduate periodontics clinic at the University of British Columbia is effective in controlling average disease progression when compared to other studies of similar duration and population characteristics.

2. Our SPT frequency is effective as well with 92% of our patients complying to their suggested maintenance schedules of 3-6 months. It is important to note however that this compliance involves those patients who returned for their maintenance visits for a minimum of 2 years and are more prone to be compliant.

3. In our well maintained population, tooth loss was generally well controlled and compared favorably to other studies.

4. Smoking has a negative impact on maintaining probing depths and attachment levels when compared to nonsmokers only during maintenance and only in deeper pockets. (≥7mm).

5. As with most studies, the majority of extractions involve molar teeth. Furcation and mobility greatly influence the long-term survivability of molars as well as the decision on assigning them with a pretreatment prognosis.

6. The results from our study show a clear difference between the 4 prognosis categories in terms of survivability at 11 years average maintenance.

7. Even when evaluating the molar tooth population only, the relationship between their survivability and prognosis was proven to be similar with our overall results. We had slightly compensated for our criteria when assigning a prognosis to
molars taking into account the impact that furcation and mobility have on tooth loss studies. It becomes apparent that this compensation is valid and should be generally implemented.

**Future Directions**

It would be interesting to see other studies using the 4-category system in assigning a prognosis and perhaps confirming our initial results. Perhaps a correlation between teeth with different prognosis may be found. A follow-up examination of our population would provide us with crucial data to further support our conclusions. Further investigation of the reasoning behind the extractions would also prove to be very valuable.

A closer evaluation of furcated and mobile molars may provide us with more information on assigning a prognosis to multirooted teeth. Their survivability was very diverse when investigating these two clinical parameters separately, but it would be beneficial to see what effect on molar survivability would furcation and mobility have when found together. This would lead to more realistic conclusions since it is uncommon to have severe furcation without mobility or vice versa in molar teeth.

The best outcome from this study would be the generation of a new prospective clinical trial focused on evaluating survivability and pretreatment prognosis assigned from calibrated clinicians.
REFERENCES


### Data from Patients Maintained Over 8 Years (n=19)

#### Appendix No. 4
<table>
<thead>
<tr>
<th>Tooth Category</th>
<th>Good</th>
<th>Fair</th>
<th>Poor</th>
<th>Hopeless</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molars</td>
<td>86.21</td>
<td>19.23</td>
<td>1.0</td>
<td>0</td>
</tr>
<tr>
<td>Premolars</td>
<td>92.31</td>
<td>5.228</td>
<td>0.572</td>
<td>0.524</td>
</tr>
<tr>
<td>Canine</td>
<td>88.24</td>
<td>13.51</td>
<td>1.0</td>
<td>0</td>
</tr>
<tr>
<td>Centrals</td>
<td>72.81</td>
<td>4.379</td>
<td>0.593</td>
<td>0</td>
</tr>
<tr>
<td>2nd Precisors</td>
<td>56.781</td>
<td>11.11</td>
<td>0.877</td>
<td>0.877</td>
</tr>
</tbody>
</table>

**Tooth Loss According to Pre-Retirement Proposals**

**Appendix No. 5**